JUNE
1942

The JOURNAL of SPEECH DISORDERS

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SCHOOL OF GRADUATE STUDIES
ANN ARBOR
INSTITUTE FOR HUMAN ADJUSTMENT

Official Publication of the American Speech Correction Association in its 16th year.

Volume 7 : Number 2

$3.00 per Year

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THE JOURNAL OF SPEECH DISORDERS is published quarterly in March, June, September, and December of each year by the American Speech Correction Association; $1.00 a copy, $3.00 a year (Canada and foreign, $3.50). All communications and subscriptions should be sent to the editorial and business office, Utah State Research Laboratories, 20th Street and Monroe Boulevard, Ogden, Utah.

Entered as second-class matter at the Post-Office in Salt Lake City, Utah, under Act of Congress of March 3, 1879.

The JOURNAL *of*
SPEECH DISORDERS

Volume 7 JUNE 1942 Number 2

THE ETIOLOGY OF STAMMERING:—

Fundamentally a Wrong Psychophysiologic Habit in the Control of the Vocal Cords for the Production of an Individual Speech Sound; A Beginning Presentation*

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Chicago

I. The Normal Activities of the Vocal Cords

In order to appreciate the action of the so-called vocal cords in the production of elemental speech sounds, we must bear in mind the following facts about them: First, acting in connection with the musculature of the chest wall, the vocal cords are primarily and normally concerned with the production of two basal involuntary and voluntary animal functions, namely abduction and adduction. Since the action of abduction opens the vocal cords, it constitutes their manner of activity for breathing; and, since the act of adduction of the vocal cords causes their complete closure, it constitutes the activity which prevents breathing. The voluntary animal uses of the acts of adduction and abduction of the vocal cords are important to life, and will be discussed in more detail in a later essay. Suffice it for present purposes to say that the larynx, through vocal cord action, serves (especially through the act of coughing), to protect the breathing organs from danger from the presence of foreign bodies. In this life-saving function the act of adduction, employed either involuntarily or voluntarily, plays a fundamental part. Both abduction and adduction of the vocal cords are employed either in coordination or independently, not only involuntarily for certain definite purposes, but also voluntarily.

*Fourth of a series of essays presenting a new conception of the etiology of stammering. The three preceding were:—

1. Peripheral Physical Inhibition of Speech; an Essential Phenomenon and an Important Causal Factor of Stammering. The Archives of Otolaryngology, December 1930, Volume 12, pp. 769-784.

2. A Critical Examination of the Foundations of the "Recoil of the Vowel" Theory of the Cause of the Impediment of the Speech in Stammering. The Journal of Speech Disorders, June 1940, Volume 5, No. 2.

3. The Etiology of Stammering; An Examination into Recent Studies; with a Glance into the Future. The Illinois Medical Journal, Volume 79, No. 4, April 1941, pp. 334-341. The above essay appears also in The Journal of Speech Disorders of March, 1941.

(This article also appears in the Illinois Medical Journal Volume 81, Number 3)

But it is to be noted that the individual, especially in this connection the child, has no knowledge of the existence of the vocal cords. Nevertheless, these organs of course are employed to accomplish both vital animal functions and eventually voice production. This lack of a knowledge of the existence and activities of the vocal cords, which, by preventing subjective knowledge of the significance of vocal cord adduction, serves as we shall see, to encourage their abnormal action in stammering.

It is also to be noted, that, through their accomplishment primarily of vital animal functions, the child's employment of voluntary abduction, as well as of adduction, definitely precedes their use in voice production. Abduction and adduction, as indicated by their involuntary as well as voluntary neurological mechanisms of control, are true animal functions, while in voice production they are employed only voluntarily, and for the especial purpose of producing sound. In the production of voice the two primary functions of abduction and adduction are simultaneously and voluntarily set into controlled opposed action so that the two vocal cords shall be held stationary and near to each other, in order to permit of being vibrated by the pressure of the breathed air.

Speech is based, first, on the psychologic creation of thought in terms of elemental speech sounds; second, on the voluntary detailed cerebral direction of the breathing function for the production of each elemental speech sound, and third, on the repetitive order of production of the individual speech sounds, for the building of words and their combination into sentences. Excepting for the various oral articulative placements required, each vocal act, or each combination of such acts, which have for their purpose the production of a speech sound, consist (perhaps more or less modified) of some factor or factors concerned with the production of the breathing function. Speech is voluntary breathing adapted to the successive production of the individual sounds of speech by the breathing mechanism.

Thus by a series of cooperating acts the entire vocal (or breathing) mechanism is concerned at one time with the production of a single elemental speech sound. The first and primary act of each of such series of acts (on which the production of each elemental speech sound depends) concerns, first, the placement of the vocal cords for production of the sound in mind, and, second, their vibration by air pressure chiefly from, but also to, the lungs, created by the action of the chest musculature in producing the impact of the breathed air against and between the vocal cords already approximated for the production of a speech sound. Thus the vibratory foundation of each elemental speech sound is produced. These basal laryngeal vibrations are modified by secondary, associated and related articulative and palatal acts.

The sounds of the environmental speech are constantly in the mind of the child, encouraging him to attempt their production, through setting into balanced opposed action the two primary animal functions of the vocal cords, abduction and adduction. As he gradually develops the capability of controlling the entire vocal machine for the production of each elemental speech sound, a psychophysiologic habit for the

creation of each sound is gradually developed. This habit also comes to involve frequently employed combinations of sounds, and also words.

II. Building the Psychophysiologic Habit of Stammering

Each animal function of the vocal cords, abduction and adduction, is voluntarily controllable. Whether (1) abduction alone, or (2) adduction alone, or (3) both together, for voice production, shall occur, depends on the voluntary visualization of a mental purpose. If (1) the mental imagery of breathing is voluntarily developed, abduction of the vocal cords occurs, resulting in an open laryngeal passage for air, if (2) the mental imagery of the complete arrest of the passage of air is conceived, the result is vocal cord adduction with complete closure of the vocal cords, and the consequent prevention of breathing and of voice production; (3) if the mental imagery of the production of a speech sound is voluntarily visualized a definite approximation of the vocal cords for that sound, due to simultaneous control over abduction and adduction, results.

Insofar as the child's mind is definitely focused on the production of a speech sound, danger of mistaken effort at voice production has no existence, and a more or less correct speech sound must result. But as the development of speech continues a danger may arise, due to the possibility of the accidental visualization by the child of the mental imagery that leads to the arrest of breathing, by which adduction of the vocal cords is produced and the production of breathing and of sound prevented. That this adduction of the vocal cords, the vocal mechanism being in action, constitutes the actual act of stammering is clearly demonstrable by intelligent observation of a large percentage of stammerers.

Indeed the key to an understanding of the act of stammering lies in the well known phenomenon of "blocking." In this typical, basal act of stammering, the musculature of the speech machine being in action in all of its parts, movement of each division of the mechanism of voice production, as well as the passage of breath, cease, while sound production is, therefore, necessarily absent. I shall be able in another essay to prove unequivocally that this phenomenon is possible only when the vocal cords are held by muscular action in a position of complete adduction.

In the development of the act of stammering, instead of focusing the mind on voice production the child becomes confused and accidentally employs not the mental imagery of voice production, but employs that for prevention of breathing, in other words, vocal cord adduction (closure). The child's mind may either quietly and unexcitedly, or excitedly and confusedly, slip into this natural psychophysiologic animal "trap." It requires a voluntary act, with which the child is more or less familiar. Even when the purpose of speech is not in the child's mind, he can accidently, or experimentally, slip voluntarily into the production of vocal cord adduction. Once he has accidentally experienced the sensations of vocal cord adduction, instead of those of voice production, his childish interest may become aroused, causing him to deliberately repeat the act. On the other hand emotional excitement

may serve as the basis for the disordered visualization that leads to vocal cord adduction. I remember a three year old white girl, who was set into violent emotional excitement by being frightened by a quite innocent act of a colored man. Exitement, weeping, screaming, and stammering began at once and the speech disorder continued into adult years. Such is one typical manner of inciting complete vocal cord closure during speech.

The onset of this interpolation of a misplaced psychophysiologic animal activity for that of voice production, which constitutes a single act of stammering, occurs almost entirely in childhood, when the normal production of voice for each speech sound is still more or less lacking as to fixity of habit action; the less the firmness of the establishment of habit for the normal laryngeal production of speech sounds, as in the earliest childhood years of speech, the greater is the tendency for stammering to take root. Until the normal habit of speech sound production has become completely established the child must direct the mind, at any rate to some degree, on the production of certain sounds or their combinations. This requirement affords the opportunity for the creation of mental confusion that may lead to the visualization of vocal cord adduction instead of that of voice production. Also circumstances of social speech situations, by creating emotion, or only mental confusion, may have the same effect.

More or less continuity, by repetition, is necessary to the development of the habit of approaching speech in this disastrous manner, if stammering is to result. For a child to start on this wrong activity but after a time to voluntarily relinquish it, is not very uncommon.

The complexities of the psychophysiologic activities of the chest-laryngeal muscular mechanism, including the subjective absence of knowledge of the vocal cords and their action, serve to create this unavoidable danger to the child in acquiring the capability of producing voice. "The trap of stammering" is created by these psychophysiologic complexities in acquiring voice, and must for all time serve to create a certain degree of danger to the child of becoming a stammerer.

III. Two Habits of Vocal Cord Action

This psychophysiologic abnormality of action of the chest-laryngeal muscular mechanism of voice production, which, after it is entered on, we call stammering, or stuttering, once well started shows exactly the same tendency to habit formation as that for normally produced speech sounds. In the stammering child, then, we eventually find two psychophysiologic habits developing simultaneously, each concerned with the movement of the vocal cords in relation to the intended voluntary production of a single speech sound; one of these developing habits is normal and competent for the production of that sound, while the other directs the organ of voice production—the vocal cords—not for the production of sound, but for the prevention of breathing and consequently also the prevention of voice production. Thus the developing child who stammers is handicapped by his growing tendency to employ the wrong

chest-laryngeal habit, rather than the correct one, which, instead of producing voice, prevents its production, and so creates at the time of stammering the impossibility of continuity of speech.

IV. The Evolution of Stammering

This unfortunate development of two habit actions concerned with the intended production of each elemental speech sound, one normal, and the other serving abnormally to prevent voice production, is subjectively not of very great importance to the stammering child, until developing intelligence serves to point out how serious his disordered speech is conceived by the people, young and old, in his environment. The sensitiveness of the mentally developing young stammerer to the smiles of people in his environment, to their mockings, and to other manners of more subtle criticism of his disturbed speech, leads directly to self-consciousness in talking. The young stammerer's sensitive self-consciousness with respect to his strangely disordered speech, very gradually becomes more pronounced, until at about ages fourteen and fifteen, when he comes to realize the seriousness of his social situation. Then the disorder begins to play havoc with the stammerer's social life, for psychologic complications necessarily are developed.

POSITION OF THE VOCAL CORDS

I.
Vocal cords abducted for breathing.

II.
Vocal cords in a balanced abducted-adducted position for production of the whispered voice.

III.
Vocal cords in a balanced abducted-adducted position for production of the loud type of voice.

IV.
Vocal cords in a state of complete adduction for prevention of breathing, — their position in the act of "blocking."

Passage from position I to position IV constitutes the act of adduction.

Passage from position IV to position I constitutes the act of abduction.

Emotion, timidity, self-consciousness, inferiority feelings and dread of attempting speech, are natural developments of his self-realization of the seriousness of his speech disorder. During speech he finds it, under such circumstances, well nigh impossible not to focus his mind on the act of stammering, instead of on that of normal voice production, which is theoretically possible. Here, again, the absence of a knowledge of the existence and activities of the vocal cords serves to prevent the stammerer from knowing what he does when he stammers and so to

conceive of protecting himself from its occurrence. Embarrassment is often constant. He knows not why he stammers or whether at any particular moment his speech will be normal or abnormal.

V. Psychophysiologic Conflict

All this psychologic social disturbance and confusion eventually leads to another definite step in the evolutionary development of stammering. A definite, but subjectively confused, psychophysiologic conflict arises, and continues so long as stammering exists. All stammerers are able at times to talk normally, and at all times it is his constant aim so to talk; but his disturbed state of mind, meaning his emotion, his impaired self-confidence, and especially his ever present fear of impaired speech, directs his mind to visualize the production of the act of stammering, and then stammer he must. The psychophysiologic conflict, then, concerns on the one hand the attempt to hold the mind to normal speech production, and, on the other, the undesired slipping of the mind into the visualization of the mental imagery of stammering. The typical stammering of the mature stammerer results from this psychophysiologic conflict. He starts to stammer, but resists by his determination to talk normally if he can, thus a seesaw physchophysiologic struggle between an attempted normal act of speech, and stammering results. Various typical behavioristic attitudes of the stammerer may also be traced to this conflict.

Largely due to the critical attitude of the public toward him, the stammerer learns to appreciate himself as a socially marked and discredited individual. He constantly faces one of the most distressing situations of any human being.

The primary explanation of stammering consists, then, not, as has been recently conceived by psychologists and psychiatrists, as some manner of general psychologic disturbance, of the individual child, but in a lurking, hidden, psychophysiologic danger involved in acquiring the control of the vocal mechanism for the production of speech sounds. It is like the loose board in the sidewalk, by which the leg may be broken.

The habit of stammering becomes deeper fixed, and usually more and more complicated, by emotion, lack of self-confidence, and fear of stammering; yet, as maturity of years and of experience produce the inevitable quieting influence on the emotions, the last years of the stammerer's life are apt to be attended with more and more success in gaining self control, and in adhering to normal speech.

VI. Proof by Treatment

With the most intelligent effort of which I am capable, I have spent many months in devising a new method of treatment, based on the conception that each act of stammering, as herein stated, represents a wrongly placed psychophysiologic habit action, which prevents the production of voice. For more than seven years I have treated stammering by this new method. The results confirm in a remarkable way the truth of the new conception of etiology.

During the ages from eight to twelve years inclusive, complete and permanent recovery has been attained in all cases so far treated. Ages eight to twelve years represent a period in the stammerer's life when, (1) sufficient intelligence on the part of the young stammerer has been attained for the employment of the required treatment, and (2) the psychophysiologic complication of emotional self-consciousness as to speech and of dread of talking, has not as yet become too seriously developed. At these early ages one is able to help the stammerer effectually to hold his mind on controlled normal talking, until the abnormal habit act of stammering has apparently actually been forgotten, or, at any rate, until normal speech has become completely dominant.

Moreover, after ages thirteen, or fourteen, the conditions and results of treatment by the new method have entirely changed. The stammerer is now mature enough to perceive that his stammering incites ridicule and criticism on the part of others. Consequently he develops serious self-consciousness as to his own talking. He is now unable to keep his mind for long away from the visualization of his habit of stammering. Therefore, one cannot teach him to talk invariably with his mind on normal, controlled speech, as can be done at ages from eight to twelve. Therefore the acts of stammering continue with more, or less, frequency, and the habit of stammering continues. Nevertheless, I do not wish to imply the impossibility of recovery during these later years, but admit only its great difficulty at the present stage of our understanding. Employing more complicated conceptions of treatment than those employed at ages eight to twelve years, and with great persistence, especially at ages beyond but near to fourteen years, complete recovery has occasionally been secured.

The attainment of marked reduction in the frequency and severity of stammering, as well as marked improvement in self-confidence, is almost always possible. Very much of course depends on the intelligence and cooperative persistence of the stammerer.

Considering the manner of treatment employed and the regularity with which complete recovery occurs at ages eight to twelve years, the reaction of the young stammerer to this form of treatment seems to me to already serve almost as final proof of the correctness of the conception of etiology herein presented.

It is also to be noted, first, that the presentation of new conceptions as to etiology of stammering have been attended in the past only with failures as to success of treatment under their guidance; and second, that the attainment of regularity of complete recovery in the treatment of stammering has never heretofore been even distantly approached at any age. And it is also to be noted that if complete recovery from stammering on a large scale can be secured during "the favorable age treatment period"—ages eight to twelve years—as now seems practically certain, a very large percentage, if not all, of stammering, may be caused to disappear after age thirteen and, fortunately, before the stammerer has suffered serious psychologic harm.

VII. Summary and Conclusions

I. The employment by the child of the chest-laryngeal mechanism of vocal cord action for purposes of speech sound production is involved in the fundamental activities of the same mechanism for animal purposes. Abduction and adduction of the vocal cords, which constitute their primary activities for animal purposes, are secondarily employed by the child for sound production. During the process of acquiring the ability to control the normal placement of the vocal cords for the production of speech sounds, certain children in attempting their production, either through sheer accident, or through emotional excitement, mistakenly visualize the mental imagery of vocal cord adduction instead of that voice production, thus producing complete vocal cord closure and preventing, instead of producing voice. If this mistaken act is repeated sufficiently, a habit of substituting the adduction of the vocal cords for that of the production of speech sounds results.

II. Thus, the stammering child develops two habits of action of the vocal cords, when it is intended to produce a speech sound, one normal for voice production, and the other that of vocal cord adduction, which prevents the production of voice. The latter constitutes the foundation of all acts of stammering.

III. As the stammering child develops sufficient intelligence to observe and interpret the critical attitude of his fellows, young and old, towards his disordered speech, he begins, at about thirteen or fourteen years of age, to appreciate its seriousness. He then comes to be much disturbed about his stammering, and develops psychologic complications: sensitiveness and self-consciousness as to his stammering, emotion, impaired social self-confidence, and dread and fear of talking.

IV. This speech situation, causing the stammerer to be seriously disturbed in social contacts, leads inevitably to a psychophysiologic conflict during speech. The stammerer desires and aims at normal speech, but his disturbed state of mind, especially emotion and fear of stammering, superinduces the visualization of the mental imagery on which vocal cord adduction depends, and he is compelled to stammer. The effort to talk normally thus is more or less constantly offset by the insistence of the habit of stammering, thus compelling disordered speech.

V. This psychophysiologic conflict, coupled with the disturbed state of mind of the stammerer, serves to explain the struggle character of the stammerer's speech, as well as its various other abnormalities, and also his typical behavioristic peculiarities.

VI. By a new method of treatment aimed to enable the stammerer to hold his mind on controlled normal production of speech, stammering, at ages eight to twelve years, can now be regularly and permanently overcome. This constitutes important evidence as to the correctness of the above conception of etiology.

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EPITOME OF AGNOSIA, APRAXIA, AND APHASIA WITH PROPOSED PHYSIOLOGIC-ANATOMIC NOMENCLATURE

J. M. Nielsen, M.D.

The nomenclature of agnosia, apraxia, and aphasia has evolved in the manner of a straggling city and for the same reason, namely that independent workers have taken different viewpoints and have become interested in one phase of the subject at a time. The terminology is in some instances anatomic, in other physiologic, in still other psychologic. Certain terms have even arisen in the minds of pedants who have constructed hypothetical diagrams on the basis of which they have invented anticipated types of clinical manifestations which were named before they were observed.

Just as grammarians arise in a nation and systematize terminology to bring intellectual order out of the chaos of human expressions, so have organizers arisen in the study of aphasia. Their efforts have been laudable but unfortunately premature because of lack of anatomic-physiologic background for their conclusions. Kinnier Wilson stated in his excellent monograph of 1926 that not only was the subject not understood but the approach was not agreed upon. He envisioned an anatomic-physiologic nomenclature as the ideal to be striven for on the grounds that a psychologic terminology was too destructive to the subject of cerebral localization to be acceptable.

Through the great revival of interest in the subject of aphasia due largely to the enormous compilation and intensive study of Henschen and the immediately subsequent invaluable contributions of neurologic surgeons, notably Foerster, Penfield, McKenzie, Gardner, German and others, so much information has accumulated during the last 20 years that Wilson's ideal seems about to be attained.

It is with considerable trepidation and fear of being considered presumptuous that I offer the present system of nomenclature. However, a study of the system will immediately make it evident that I am presenting exceedingly little by way of original terminology; I am rather organizing present knowledge. The terms agnosia, apraxia, and aphasia can be retained with slightly modified definitions. The greatest single change suggested is one of the concept of the function of the minor cerebral hemisphere in language. Even this concept is far from new as it was suggested by that great master Hughlings Jackson and elaborated considerably by Henschen. Potzl's work in localization and Kleist's intensive studies are landmarks worthy of careful study.

The Functions of the Minor Cerebral Hemisphere

It is customary to speak of the two cerebral hemispheres as being dominant and recessive, respectively. The terms are based on the conception that the one ordinarily takes the lead and leaves little or nothing

for the other to do in the associative sphere of activity. It is tacitly understood that dominance does not apply to projection fiber systems or to the basal ganglia.

The term *dominance* or *dominant hemisphere* is expressive but inaccurate. The studies of many excellent authors on thousands of cases have shown that the relation of the so-called dominant hemisphere to the recessive is such that it can be much more accurately represented by the relationship of pilot and co-pilot on an airliner. In the beginning of their associated careers the pilot is slightly more expert than the co-pilot, but in case of sudden incapacity of the pilot the co-pilot can manage the ship almost equally well. The pilot does not dominate the co-pilot. I have suggested the use of the terms major and minor hemispheres as being more appropriate than dominant and recessive hemispheres because they suggest merely "more" and "less." The terms major and minor are already in wide use in industrial medicine relative to hands, feet and eyes.

There is, however, an important discrepancy between the two relationships in our simile (the pilot-co-pilot relationship and the two-hemisphere relationship) which must be clarified. The pilot ordinarily turns over the management of the ship to the co-pilot during at least a short portion of each flight and the co-pilot therefore remains proficient. The major hemisphere ordinarily never turns over its functions to the minor but instead becomes more and more proficient while the minor misses opportunity for further training. It is capable, however, of rapid training when the necessity arises if the patient (and the minor hemisphere) are not too old and if they are in good general condition.

So far as the *language* function of the brain is concerned, another simile is much more accurate. Consider a hypothetical situation of a group of men traveling together through a foreign country. Only one, the leader, has studied the language of the foreign country and is expert in it. The followers have not studied it. The group travels for some months, when suddenly the leader is killed. The proficiency of the followers to understand and speak for themselves will depend entirely on their individual independence and initiative. Some followers will have learned nothing, some will have acquired a short vocabulary and will have learned to understand a few simple phrases. Others will have read, listened, and spoken for practice with a view to preparedness. A very few will have become proficient.

That is the case with the major and minor cerebral hemispheres. They start through the world together almost equally incompetent. In some instances, when the major is destroyed the minor hemisphere will have developed its engrams so crudely that it does not function at all. In other instances it will be able to formulate language slightly, understand a little immediately and improve rapidly with training. In a very few instances it will have established its engrams so well that when the major hemisphere is suddenly destroyed, it will be able to assume the functions of language.

When, in the above simile, minor cerebral hemispheres were compared with individuals they were actually compared with major cerebral hemispheres, because the individuals in the illustrations were using

their major hemispheres. While there is a hereditary determinant to make a certain hemisphere the major one, there is no essential difference between the functional capacity of major and minor hemispheres at birth. This has been shown repeatedly by instances of severe trauma to the major hemisphere early in life. In such cases the minor hemisphere invariably takes over the function of the major with such precision and dexterity that observers do not notice any disturbance of language. Handedness does not develop until the age of about nine months to a year. Even when it begins to manifest itself the superiority of the major does not appear great. This is proved by the fact that injury to the major hemisphere at the age of four or five years causes only transient aphasia. Whenever a minor hemisphere is called upon to take over a function it at first fatigues with astonishing rapidity. As it becomes trained it develops endurance rather than specific capacity.

"Majority" and Handedness

The common hereditary tendency to right-handedness is actually a tendency to left-brainedness; the brainedness comes first. The handedness can be shifted by training early in life but the brainedness, as a unit, does not shift with it. Well-meaning mentors frequently change left-handed individuals to right-handedness at so early a time in life that the subject may not be aware of it at all. This accounts for some instances of right-brainedness in "right-handed" persons. Aside from these occasions, however, there are instances of left-brainedness in left-handed persons. Ipsilaterality of handedness and brainedness occurs, according to the figures of Chesher, in about six per cent of all persons. Hereditary right-brainedness, as shown by the fallible index of left-handedness, occurs in about ten per cent of all persons. Because of these discrepancies between handedness and brainedness it is never safe to lateralize a cerebral lesion on the basis of aphasia alone. Unequivocal lateralizing signs based on projection fiber systems must be relied upon.

Up to this point, to facilitate clarity, the discussion of function of the minor cerebral hemisphere has centered about it as though it were a unit. Extensive research, however, has shown that the following subdivisions must be considered separately:

(1) Prefrontal lobes.
(2) The frontal writing center (foot of the second frontal convolution).
(3) Broca's convolution.
(4) Pars triangularis of the third frontal convolution.
(5) Anterior end of the superior temporal convolution.
(6) Wernicke's area (posterior third of the superior temporal convolution).
(7) Area 37 of Brodmann.
(8) Angular gyrus (area 39 of Brodmann).
(9) Area 18 of Brodmann.
(10) Area 19 of Brodmann.
(11) Convolutions of Gratiolet.

These eleven areas are all association areas, i.e. they are not areas of cortex concerned immediately with reception or transmission of impulses over the projection fiber tracts. The eleven areas occur in pairs, of course, but the degree to which the major in each case is superior to the minor is not the same for all pairs. Disregarding for the present the exceptional cases one may state the case for each area in the average person as follows:

The prefrontal lobes, so far as their intellectual functions are concerned, either establish no unilateral superiority or establish it to a small degree. They seem at times to divide the various functions of the lobes between them, this being manifest by the appearance of certain symptoms regardless of which frontal lobe is affected. But in any case either right or left prefrontal lobe seems capable of assuming the necessary function of both after a short period of training (1).

Area 18 of Brodmann (area parastriata) is concerned with recognition of objects and pictures. The evidence available at present indicates strongly that either the right or the left becomes the major during the first year of life and that the selection is purely a matter of chance. In most persons the degree to which either becomes the major is relatively slight and destruction of the major one leads to only temporary disability of recognition of objects. In a few persons (relatively speaking) the degree of superiority of one becomes so great that its destruction leads to visual agnosia for objects lasting for years. In most instances the major area 18 is ipsilateral to the major temporal lobe (when the major temporal lobe is determined by language function). In a few instances the right remains the major because of an early fixation even though the major temporal lobe becomes established on the left side (2).

Area 19 of Brodmann (area peristriata) is concerned with revisualization of former images. It follows the rules of lateral superiority described as governing area 18 (2).

The three groups described (prefrontal lobe, area 18 and area 19) are not concerned with language and this fact probably underlies the weakness of unilateral superiority. There is much evidence that the superiority of the occipital lobes actually shifts, when it does shift, with the establishment of language centers (second year of life). The lowest function which can be called language is music. It will be seen as we proceed that the areas concerned with vocal, instrumental, and auditory music perform their function almost equally well on the two sides.

The *pars triangularis of the third frontal convolution* (area 45 of Brodmann) is concerned with all of the functions of vocal music and the playing of musical instruments. Injury to it causes apraxia for these acts if it causes any symptom. But it has been abundantly verified that the minor side when called upon takes over the function with great ease and that in only rare instances is there any functional defect after injury to only one side (2c).

The *anterior extremity of the superior temporal convolution* (area 38 of Brodmann) is concerned with musical auditory recognition and interpretation. It receives impulses from the transverse gyri of Heschl which act as centers of primary auditory perception. Destruction of

area 38 of either side usually produces no disturbance of musical comprehension because the other side takes over the function. Destruction of the area bilaterally destroys the auditory musical sense (2c).

Wernicke's area (areas 41 and 42 of Brodmann) is concerned with interpretation of spoken language. Comprehension of spoken language is next in scale above comprehension of music and in harmony with this fact the degree to which the major side ranks above the minor is next in the scale. After removal of the major temporal lobe the average patient is able to comprehend about five or six questions addressed to him. He (his minor temporal lobe) then fatigues. After a year he usually understands a great deal of what is said and after two years one can as a rule hardly tell in ordinary conversation that anything has been removed. The right has assumed the function (3).

Area 37 of Brodmann is concerned with formulation of language. This function is really the one which has been called internal language. Destruction of area 37, or in some cases even relatively slight damage to it, results in inability of the patient to find his words and formulate his sentences. The degree to which the major side is superior is almost identical with that described for the area of Wernicke (4).

Formulation of language is a complex cerebral function dependent on several subsidiary functions. An area of cerebral cortex essentially identical in extent with area 37 of Brodmann is of the utmost importance for formulation of language, either written or spoken. Difficulty with word finding occurs in some cases of angular gyrus lesions, in perhaps one-half of the instances. The same difficulty occurs in a few instances of lesion of the posterior portion of the superior temporal convolution, i.e. when the lesion is behind Wernicke's area. Lesions of Wernicke's area itself interfere with formulation of language to such an extent (as a rule) that the patient does not speak.

These facts explain the views of Kleist that reauditorization of a word is necessary to formulation of language. He calls area 37 of Brodmann an acoustic-psychic sphere.

Kleist states that the word sound is not simply apperceived acoustically and then immediately connected with the concept of the object, but it first appears in consciousness. He says that something is meant with that speech sound, that it designates something without the individual's becoming conscious of what specific object is meant. While all this is true, there is nevertheless much more to the determination of the meaning of a word than the process described above. Some words cannot be distinguished by their sound alone or by their appearance alone. Some words alter their meaning according to context. The expression "they read" cannot be pronounced until context is added. We need to know whether the tense of the verb is present or past. And one cannot distinguish between "red" and "read" by the sound alone. The endless punning heard in entertainment is possible because of these peculiarities (imperfections) of language.

Because of these facts I cannot conceive of area 37 of Brodmann as being merely an acoustic-psychic sphere. It is an area in which knowledge is coordinated for significance of words and expressions and in which grammar, rhetoric, and syntax are considered in the formulation

of language. The area is in constant association with all of the other areas concerned with language and its separation from the other areas by organic lesions may give rise to defects of formulation of language.

Lesions of this area provoke as their most constant symptom amnesic aphasia. The area for formulation of language is chiefly area 37 of Brodmann but this area is so trained by association with Broca's convolution, Wernicke's area, and the angular gyrus that in some instances a lesion of one of these cripples the function of area 37 sufficiently to cause it to "resign" and turn over the function of formulation of language to the identical area of the minor side. The result is amnesic aphasia. I believe that this disturbance should be called formulation aphasia. (The term "formulation" need not be negative so long as it is followed by "aphasia.") Whether misuse of words, use of wrong words (commonly called paraphasia), or the more severe forms called jargon aphasia or agramatism result depends on the degree to which the minor side takes up the function. The minor side is usually capable of assuming to some degree the function of formulation of language, but, until it has had time for training, it functions imperfectly. The most pronounced defect is seen in loss of ability to associate objects and their names.

Broca's convolution contains engrams representing the physiologic basis of memory of how to use the vocal organs for the production of words. These engrams are formed simultaneously with, or very shortly after, those of Wernicke's area. The major is superior to the minor in about the same degree as already stated for Wernicke's area, i.e. far superior. Destruction of it in adulthood leaves the patient, in the great majority of cases, able to say only a few words, which he uses for all responses. Usually several years are required for training of the minor side; but as a rule, if the general cerebral function is of good quality, the minor side becomes nearly as proficient as the major had been (8).

The convolutions of Gratiolet, located between the angular gyrus and the second occipital convolution (zone between areas 19 and 39 of Brodmann) are concerned with the body scheme. As the naming of parts of the body, especially naming of fingers and designation of laterality, includes phases of language, disturbances of the body scheme follow the rules for language areas rather than those for the non-language areas regarding laterality. The major is superior to about the degree described for the area of Wernicke, i.e. greatly superior, as the syndrome occurs with great regularity from lesions of the major side (5).

The frontal writing center and the *angular gyrus* are concerned with the most highly specialized of language functions, emission and reception, respectively, of written and printed language. The writing center at the foot of the second frontal convolution is essential to the memory of handwriting movements. In the majority of cases it cannot function without close association with the convolution of Broca. It cannot, of course, function in writing unless the language formulation area (area 37 of Brodmann) and the area of revisualization of the written word (angular gyrus) are intact and in communication with it. The major writing center is far superior to the minor in most cases. A long period of training is usually necessary for the minor to function

at all well. Unless the destruction of the major occurs early in life, the minor rarely attains a degree of perfection comparable to that which the major had enjoyed. Conversion of a left-handed person to right-handedness for writing establishes a writing center on the minor side (6).

The *angular gyrus* has a double function. Its cortex contains patterns of two types, one for recognition, the other for revisualization of the written and printed word. The patterns of both types are spread diffusely over the cortex and the superiority of the major is great. The minor angular gyrus in most instances has its engrams so crudely made that many years are required, after destruction of the major one in adulthood, for it to perform at all well. Unless the major is destroyed early in life, the minor rarely attains a degree of perfection comparable to that of the major.

The angular gyrus is trained in very different ways in different persons and consequently its degree of ultimate independence varies enormously. When a child learns to read he at first associates very intimately the engrams for the enunciated word (for the word spoken by himself) with the auditory memory engram of the word which he reads. This is obvious when one recalls that the child reads aloud; he *says* the word and he *hears* himself say it. During the formative period this association of the engrams of the angular gyrus with those of Broca's convolution is so intimate that a lesion of Broca's convolution prevents the child from even *recognizing* the written or printed word. If his training in reading does not advance beyond the point of enunciating (or at least retaining a habit of lip movements while reading) the angular gyrus never becomes independent of Broca's convolution. If the child is taught to read by simultaneous writing of the word the angular gryus may become and may remain dependent on the frontal writing center also. In such cases alexia results later in life from a frontal lesion. However, in the majority of educated persons the angular gryus develops, by silent reading, an essential independence of the centers in the frontal lobe.

The situation is somewhat different regarding the continued dependence of the angular gyrus function on the area of Wernicke. In the area of Wernicke one "auditorizes" the word one is reading and this habit in most instances continues throughout life. For this reason, at any time of life,, in the majority of cases, destruction of Wernicke's area, or destruction of the short cortical area between the angular gyrus and Wernicke's area, produces alexia by preventing not only interpretation but also recognition of the written or printed word. It is because of the elaborateness of the angular gyrus connections that the major becomes so strongly superior to the minor and that training of the minor, when the necessity arises, is extremely difficult (7).

In addition to these important facts about the functional capacity of the various cortical areas of the minor side there is another principle which has not been enunciated, but which is becoming more and more evident and which is of the utmost importance to the understanding of localization in agnosia, apraxia, and aphasia. This is the principle that a given area of cortex concerned with language may, after a

minor lesion in it, either attempt to carry on its function, though crippled, or resign immediately. The question whether it continues to function after it is crippled or resigns depends on the facility with which the corresponding minor area can assume the function. Thus a strongly superior language formulation area (area 37 of Brodmann) may continue to function, though imperfectly, after a minor lesion, while the same area in another case with a similar lesion may resign and turn over the function to the minor area. There is no absolute criterion by which an examiner can determine which has happened in a given case. In the majority of cases the damaged area resigns its job. It is for this reason that the brain of a person who has been unable to formulate his sentences may show a tiny lesion in the area concerned or it may show a complete destruction of the area. The whole matter resolves itself into this question, "Can the crippled major area perform with greater ease than the normal but untrained minor area?" The individual in question will take the easiest course. With these facts before us we can see that a positive agnostic or aphasic symptom has a positive value in diagnosis but a negative one has little if any value. If the projection fiber signs and symptoms indicate that an area concerned with aphasia is destroyed, but there is no aphasia, the negative finding of absence of aphasia has little if any weight in determining the extent of the lesion.

We now come to the question, "How complete must the defect be to constitute agnosia or aphasia?" If, following a lesion of the convolution of Broca, a person is able to speak but with stammering and hesitancy, some students might say that he does not have Broca's aphasia. But we know that from the standpoint of cerebral localization he does have a lesion and that the reason for his being able to talk at all is either that the crippled area is performing or the imperfectly trained minor convolution of Broca is doing the work which we observe. We should therefore say that the patient does have aphasia (apraxia of speech). If the patient, after such a lesion, speaks as well as he did before he either had two equally capable convolutions of Broca or it was the minor one which was destroyed, the minor one merely having been located on the side where we expected the major one to be. Our misinterpretation is due to the erroneous assumption that handedness is an infallible guide to brainedness.

We thus arrive at the principle that a disturbed function of language has the same weight in cerebral localization as a complete loss of the function. A corollary of this is that in the study of human cerebral function in the associative field (by the use of clinicopathologic material to which we are limited) it is impossible to determine the function of any area unless we confine our studies to *bilateral* lesions of the area in question. If this principle had been recognized earlier, millions of words could have been omitted from the scientific literature on aphasia and allied branches. If a given area is bilaterally destroyed we are certain of a functional defect. The determination of the functional defect by a clinical study is not difficult in the light of present knowledge.

Apraxia

The function of the minor cerebral hemisphere in acts not pertaining to language is far greater than in language functions because handedness and brainedness are largely governed by the development of language. It is for this reason that apraxia is far rarer in the non-language sphere than in the apractic aphasias.

In the precentral gyrus are located patterns of movement, physiologic patterns for coordinated motor performance. Physiologically speaking, on a par with these patterns are many others. Loss of function of these patterns has been called kinetic apraxia of the limb (limb-kinetic apraxia of Liepmann). I have suggested the term cortical motor pattern apraxia because in many instances the limbs are not involved. Illustrations of this type of apraxia are: Broca's aphasia, dysarthria and dysphagia resulting from a lesion of the precentral gyrus of the mouth area, paralysis of voluntary lateral gaze with preserved reflex lateral gaze resulting from a lesion of the foot of the second frontal convolution, apractic agraphia resulting from a lesion of the frontal writing center, loss of finger movement with preservation of use of the upper limb as a whole resulting from a lesion of the precentral gyrus, avocalia (of Henschen) from a lesion of the pars triangularis of the third frontal convolution, and probably a few other instances of disorganization of movement resulting from lesions of areas 6-a and 5-a. In all forms pertaining to language unilateral dominance is relatively strong; in the others dominance in most persons plays a minor role.

These various centers of performance of *movements* are in turn governed for the performance of *acts*. An arbitrary differentiation must be made between movements and acts, the latter consisting of a series of the former coordinated to the carrying out of a plan of action. There is no "center" for carrying out an act. Loss of the power to do so (in the absence of paralysis, ataxia, or dementia) is ideokinetic apraxia. Lesions of the corpus callosum or deep lesions of the major supramarginal gyrus are most potent but in any case we are dealing with a disturbance of associative function and not a cortical lesion.

There is relatively slight dominance of one hemisphere over the other in ideokinetic apraxia for which reason the condition is rare relative to aphasia and usually transient. Surgical severance of the corpus callosum does not cause apraxia. The ideational plan of action can usually be formed in either hemisphere for both lateral sets of limbs. Ideokinetic apraxia represents physiologically an interruption between the ideational plan and the patterns for execution of an act.

When the ideational plan itself is disturbed, i.e. when an ideational plan cannot be kept in consciousness long enough for its execution, the condition is called ideational apraxia. This condition is always due to a diffuse cerebral lesion, quite often a toxemia.

Selection of Terms

The reasons for the choice of each term will be much clarified by development of the physiology in brief and simple language. The function of area striata has been designated as *primary perception*. While it is true that there is no perception in the usual sense until the impulse

reaches consciousness, and since the visual impression does not reach consciousness until impulses reach the diencephalon, the area striata does not really perform the function of perception. However, common usage has dignified the term primary perception, and it seems wise to keep it.

So far as the cortex is concerned, the next physiologic step above the level of primary perception is recognition and for this function area 18 of Brodmann is essential. Loss of the function of recognition resulting from an organic cerebral lesion and through one sense organ only is *agnosia,* and a distinction must be made between agnosias for each of three senses; olfactory and gustatory agnosias are not definitely known. Loss of the function of visual recognition is *visual agnosia.* From the standpoint of cerebral localization it is necessary to distinguish between recognition of animate and inanimate objects because one function is not infrequently lost without the other. And as objects have many attributes such as form, color, distance (dimension), and direction and as each of these attributes has been known to be lost separately through an organic lesion of area 18 (parastriata) we are compelled to acknowledge *general visual object agnosia, visual animate object agnosia, visual inanimate object agnosia, visual form agnosia, color agnosia, visual distance agnosia, and visual direction agnosia.* In the realm of animate objects, organic cerebral lesions cause agnosia separately for certain parts of the body, especially the fingers. We thus have *visual finger agnosia.* Visual agnosia for the entire body has occurred from a cerebral lesion and this has been described as autotopagnosia (referring to agnosia for topography of oneself). The term, however, should be qualified by the adjective visual, making *visual autotopagnosia.* The lesion causing this condition is found in the occipital lobe, areas 18 and 19 of Brodmann of the major side, or both sides. All visual agnosias not concerned with language result from occipital lesions, but in order to have a consistent nomenclature an anatomic term must be added to each.

The localization of the lesions causing agnosias *in the sphere of language* is not so simple. The area of cortex essential to visual recognition of letters, syllables, words, and musical notes is in the angular gyrus. The similar area for mathematical figures is made up of the borders of the interparietal sulcus. (One little exception to these statements which must be made because of its importance in treatment of certain cases is this: when the infant first learns letters they are to it not symbols but objects. They are therefore stored as engrams in area 18 of Brodmann at that time. They may remain there ready for use and this explains the fact that adults who have had their angular gyri destroyed and who consequently are totally unable to recognize syllables or words, sometimes recognize letters and are thus able to spell words aloud and recognize them by their sounds.) Visual verbal agnosia, visual literal agnosia, visual numeral agnosia, and visual musical agnosia all result from cortical destruction of the corresponding cortical areas. But this is not the only possible site for the causative lesions because subcortical destruction in the same area or between the cortical areas in question and the occipital pole may cause it also.

Moreover, in the cases in which the individual affected has established strong associations between the angular gyrus and Wernicke's area (which is the rule) or between the angular gyrus and the convolution of Broca (the exception) destruction of these associated areas may cause visual agnosia for letters, words, etc. In a given case one cannot tell by the agnosia alone where the lesion is. Other findings in the case give the clue. Thus hemianopia would indicate temporal, lower parietal, or occipital location; hemisparesis would indicate a capsular or frontal cortical lesion, etc. Because of these possibilities anatomic terms in the form of adjectives must be added to the physiologic descriptive term in designating the agnosia.

We thus have *visual parietal numeral agnosia, visual occipital numeral agnosia, visual angular literal agnosia, visual occipital literal agnosia, visual temporal literal agnosia, visual insular literal agnosia (rare), visual frontal literal agnosia (rare), visual angular verbal agnosia, visual occipital verbal agnosia, visual temporal verbal agnosia, visual insular verbal agnosia, visual frontal verbal agnosia, visual subcortical angular musical agnosia, visual musical angular agnosia, visual musical occipital agnosia, and visual subcortical angular verbal agnosia.* A corresponding series with the word *syllabic* substituted for *verbal* could be made but is hardly necessary. The distinctions between cortical and subcortical lesions of the angular gyrus with reference to agnosia are made clinically by the occurrence of simultaneous agraphia in the former (cortical) and its absence in the latter.

The auditory agnosias all result from temporal lesions. The reason that temporal lesions can cause visual verbal agnosia while angular gyrus lesions do not cause auditory verbal agnosia rests on the fact that the temporal lobe is trained in the infant and child for four to five years before the angular gyrus comes into function. The angular gyrus is usually dependent on the area of Wernicke but the area of Wernicke is independent of the angular gyrus.

Of auditory agnosias we have *general auditory temporal agnosia, auditory temporal verbal agnosia,* and *auditory temporal musical agnosia.* Auditory verbal agnosia results from a lesion of Wernicke's area and occasionally from lesions in its immediate vicinity within the temporal lobe because in some cases the pressure or edema from a neighboring lesion causes the area of Wernicke to "resign." Auditory musical agnosia is rare because the minor side assumes the function easily. When it does occur it results from a lesion of the anterior extremity of the superior temporal convolution (area 38 of Brodmann). It is entirely useless to consider the site of the causative lesion in transcortical or subcortical sensory aphasia because the lesion is located in a position identical with that for ordinary auditory verbal agnosia. The same statement applies to paraphasia. Paraphasia or senseless repetition of sounds heard is simply due to imperfect performance of the minor side.

Olfactory and gustatory agnosias are still impossible of diagnosis either clinically or pathologically. It is doubtful whether they occur.

Tactile agnosia (astereognosis) may result from a lesion of the parietal lobe (usual) and very rarely from an occipital lesion. In the

former case the *tactile parietal agnosia* is unilateral only and contralateral to a lesion of the postcentral gyrus, superior parietal lobule, or supramarginal gyrus. It is probable that the postcentral gyrus is the essential area concerned and that lesions in the other two areas cause tactile agnosia by edema or pressure. When tactile agnosia results from an occipital lesion it is invariably associated with a lesion of area 19 of Brodmann. The patient is unable to form visual concepts of objects by either the visual or tactile route and hence the tactile agnosia (astereognosis) is bilateral even from a unilateral occipital lesion. Such a clinical condition is now known as apperceptive blindness of the senile (Pick) but should be called *occipital tactile and visual agnosias*. This term indicates that the area for revisualization of former images is out of function and its removal is causative of the tactile agnosia (there are two simultaneous agnosias).

When the body scheme is disturbed (by a lesion of the gyri of Gratiolet) the patient may have separate tactile agnosia for the fingers. This should be called *tactile finger parieto-occipital agnosia*. If the entire body scheme is disturbed, *visual autotopagnosia* results.

In the use of the term agnosia, there is only one slight modification of its past usage that needs to be agreed upon, namely that agnosia should apply in any case to recognition from one sense organ only. Its misuse in the past has resulted from inaccurate thinking. Several unacceptable terms have thus arisen. We cannot logically speak of ideational agnosia because ideation goes far beyond recognition. Neither can we speak of loss of finger recognition as finger agnosia unless we mean either visual recognition or tactile recognition. If both are present we have two simultatneous agnosias before us.

The third step in sensory cortical function is that of reactivation of former engrams (images in the visual sphere, sounds in the auditory sphere, tactile reconstructions in the tactile sphere). This third step consists in bringing into consciousness memory from the past. We have been accustomed to speak of it as revisualization in the visual sphere and as recall in the other two spheres. The term recall is distinctly English and not at all specific for the function of cerebral function. The term amnesia is in universal use for forgetting, but forgetting is often a positive process, not merely a failure to remember but a relegation of something unpleasant to the subconscious. Moreover, all agnosias, apraxias, and aphasias are forms of amnesia. There is an international term which means the process of reliving a former memory, namely reminiscence. The failure to effect *reminiscence* could be termed *irreminiscence* and the verb, now only colloquial in English, *reminisce*, can be dignified with usage. I therefore suggest that the function of the tertiary sensory cortex be called reminiscence. We can then speak of *visual, auditory, tactile, olfactory*, and *gustatory reminiscence* and *irreminiscence*.

The function of the tertiary visual cortex (area 19 of Brodmann) is, then, that of visual reminiscence. By means of area 19 (area peristriata) one relives former visual memories. It must be recalled that area 18 is the servant of area 17 and functions only through it (area 18 serves the purpose of recognizing images primarily perceived by

area 17) and one cannot voluntarily stimulate it without allowing area 17 to function again by the patient's voluntary act of looking again. But area 19 is the card index to former memories in the service of all the rest of the brain. One can revive a former image by hearing an associated sound, by feeling an object which is associated in memory, by smelling or tasting a substance which can revive the memory or by seeing an object which has associated memories. But if one reactivates the engrams of area 19 by seeing, one works through area 18. The other four senses reactivate area 19 directly. Its reactivation is reminiscence.

For verbal or musical visual symbols the angular gyrus serves for reminiscence. For numerals the borders of the interparietal sulcus serve the purpose. For reminiscence of the body scheme the gyri of Gratiolet are essential.

Visual irreminiscence is manifest clinically by loss of power to revisualize objects and all attributes of objects such as form, color, direction, dimension and relative distances between objects or parts of objects. Loss of power to revisualize cardinal directions, street scenes, one's home, one's friends or one's body have all been observed following lesions of area 19 of Brodmann. We thus have: *form irreminiscence, visual occipital; color irreminiscence, occipital; distance irreminiscence, occipital; direction irreminiscence, occipital; visual verbal irreminiscence, angular; visual musical irreminiscence, angular; visual literal irreminiscence, angular; visual numeral irreminiscence, parietal; visual finger irreminiscence, parieto-occipital;* and when the body scheme is affected, *visual body irreminiscence, parieto-occipital.*

In the auditory sphere cortical areas concerned with reminiscence cannot be sharply separated from the areas for recognition. It seems that the portion of temporal cortex juxtaposed to the musical and general auditory cortices for recognition serve for reminiscence. It is probable that the area essential to the function varies considerably in different individuals. Clinically it is well known that *auditory musical irreminiscence, temporal; auditory verbal irreminiscence, temporal;* and *general auditory irreminiscence, temporal* occur. One may be unable, after a temporal lesion, to recall a tune but able to recognize it when heard, or one may lose the exact wording of a quotation while retaining a concept of the idea expressed and recognize the words when they are spoken.

Aphasia has been defined in the past as loss of the power to comprehend or express signs and symbols by which man communicates with his peers. It has been agreed that dementia, psychotic disturbances and dysarthria are excluded as causes and that aphasia is always due to an organic lesion of the brain. We can smplify this definition somewhat by saying that *aphasia consists in organic disease of the cerebral memory engrams for language.* Either of these definitions makes aphasia embrace those agnosias and apraxias which have to do with language. I suggest that in any compound term embodying the word agnosia or apraxia the added term of aphasia is a redundancy and not advisable. We can reserve the use of the term aphasia in our nomenclature for designating "the loss of language association with." Thus *visual finger*

aphasia means loss of power to associate the written terms for the fingers with the *object* (finger), *color aphasia* loss of power to associate the language symbol with the color. When one wishes to distinguish between the loss of power to associate the symbol and the loss of power to determine the *significance* of the symbol in terms of language, the adjective *semantic* introduced by Head serves nicely.

There are, of course, all degrees of semantic aphasia even within the present use of the term. So simple a term as the verb *to read* may have a great variety of significances. It may mean:

a. To pronounce the sound indicated by the written symbol without even the faintest understanding, just as I may "read" Welsh (of which I am ignorant).

b. To render the symbol in another language, as to say that this P is an R because the language is Greek.

c. To interpret the word symbol in its most elementary way as to say that that word (cat) means an animal like the one we have in the house.

d. To interpret the symbol in any degree of complexity as to say that "cat" means a domesticated, carnivorous quadruped of the feline family, or to interpret many pages of a legal citation as blackmail, or to interpret the scriptures. To read has even wider meanings as in "to read between the lines" or to read a latent content into a dream.

However, if we are to establish a practical nomenclature such a range of semantics must be discounted. We must be willing to accept an arbitrary distinction between simple recognition and recognition plus interpretation.

It will be necessary to distinguish carefully between the object and its symbol. Loss of *recognition* of the object or its symbol is agnosia for the object or the symbol. Loss of *significance* of the symbol is aphasia.

Inasmuch as all of the old motor aphasias are forms of apraxia it will be an improvement to call them apraxias, or apractic aphasias.

With these explanations we can proceed to elucidation of terms. It is practical to work out from the known agnosias to the aphasias, both anatomically and clinically.

An object may be recognized and comprehended without the patient's being able to establish the symbolic association by seeing or feeling the object. The patient says, "I know what it is but I can't name it." This is the classic amnesic aphasia. Inasmuch as the difficulty is one of language with reference to the object, this is properly called visual object aphasia and tactile object aphasia. In either case the lesion affects the association between the occipital area for visual recognition and the temporo-angular-formulation area or the association between the parietal lobe and the temporo-angular-formulation area. This area is in the posterior part of the temporal lobe, especially area 37 of Brodmann. Hence we derive the terms *visual temporal object aphasia* or *tactile temporal object aphasia* and both are usually present in the same case. The same syndrome applied to colors need not be encumbered with the adjective visual since colors are recognized only

by vision. We therefore select the term color aphasia but as the lesion may be occipital or temporal, we have *temporal color aphasia* and *occipital color aphasia.* Loss of ability to identify fingers by name is exactly parallel and is *visual finger temporo-occipital aphasia.* In this defect we have two categories, *visual temporo-occipital finger aphasia* and *tactile parieto-occipital finger aphasia.* When other parts of the body are affected the part may be named accordingly.

Gustatory and olfactory aphasias are at present unknown.

Auditory aphasia consists in the loss of ability to associate the name with the sound of an object such as loss of ability to name a typewriter by hearing its sound. It practically always accompanies auditory verbal agnosia, only one case having been reported in which it occurred separately. *Auditory musical aphasia* if it ever occurs is extremely rare. In auditory aphasias the lesion is always in the temporal lobe.

Semantic aphasia is aphasia of significance. It does not come into play relative to objects because by the time one has recognized the object, and has named it, its significance is known. Thus if one recognizes the sound of a saw and is able to name the object the significance is complete. This is not true with reference to language symbols. One may see a certain word or phrase and be able to select from a given copy all similar words or phrases and still be totally unaware of their significance. Or one may hear and recognize a word (as shown by ability to repeat it) and still be totally unaware of its significance. Such defects are *semantic aphasias.* The visual semantic aphasias may be described as alexias but they are still aphasias.

We have *visual semantic external capsular aphasia* (loss of significance of written or printed matter through removal of the practic component), *visual semantic temporal aphasia* (loss of significance of written or printed matter due to a lesion between the angular gyrus and Wernick's area or in Wernicke's area itself), *visual semantic third frontal aphasia* (loss of significance of written or printed matter from destruction of the practic component), *auditory semantic temporal verbal aphasia* (loss of ability to determine significance of spoken language though able to recognize it as spoken language), and *auditory semantic temporal musical aphasia* (loss of ability to determine significance of the music though able to recognize it, type of auditory amusia).

There is also *formulation aphasia* or inability to formulate language (the old paraphasia or jargon aphasia).

The Apractic Aphasias

Inasmuch as the types of aphasia which have been called "motor" are all forms of apraxia and as apraxia is only one form of motor disturbance the term apractic aphasia is far more exact than "motor aphasia." It is necessary to retain the two designations "apractic" and "aphasia" because some apraxias do not concern language at all.

The motor patterns located in the convolution of Broca are engrams of the memory of how to make certain sounds with the organs of speech. Disease of these engrams causes motor pattern apraxia (the type which Liepmann called kinetic apraxia of the limb but which in

this instance does not affect a limb). As the impulses from these cortical engrams must reach the precentral gyrus in order to produce speech, it is feasible for a lesion between Broca's convolution and the precentral gyrus to render speech impossible. It is also possible for a lesion of the anterior portion of the insula to extend deeply enough (a few millimeters to a centimeter into its substance) to give the same syndrome.

Beside these mechanisms which can render speech impossible, there is the mechanism of preventing formulation of language. Lesions at the temporal isthmus, by destroying the cross-road of association fibers, can so interfere with language reminiscence that the patient has nothing to say, i.e. he has no speech ideation. For this reason when the general physical examination indicates a severe posterior temporal lesion and the patient is also speechless, it is not necessary to assume an apraxia of speech. The patient must have ability to formulate his language before he can send language-composition impulses to the motor area and the motor area must not be blamed for failure to dispatch impulses which do not reach it.

The apractic aphasias are, then, *frontal apractic aphasia* (the old Broca's aphasia), *insular apractic aphasia, frontal triangularis apractic amusia* (lesion of pars triangularis, which contains engrams for praxia of music), *second frontal apractic agraphia* (due to lesion of Exner's writing center at the second frontal convolution), and *third frontal apractic agraphia* (the agraphia accompanying apractic aphasia).

Apraxia

The physiologic basis of the apraxias has already been discussed. It has also been pointed out that the motor aphasias, some types of agraphia, some disturbances of conjugate lateral turning of the eyes and some cases of dysarthria and dysphagia, etc., are types of apraxia. There is no difference between apractic amusia and musical apraxia, or between apractic aphasia and aphasic apraxia; the selection of the term in a given case of that sort depends on one's viewpoint at the moment of discussing the subject. One point, however, must remain clear, namely that agnostic apraxia is an impossibility because apraxia based on agnosia is a form of agnosia. There cannot be a sensory apraxia of any sort. But we may have the reverse, apractic agnosia which is an agnosia resulting from destruction of a motor pattern (an apraxia) which prevents the sensory pattern from functioning. In such cases the anatomic term will clarify the idea in each case.

The apraxias will therefore embrace: *apraxia* (for any particular act or in general), *cortical motor pattern apraxia, ideokinetic callosal apraxia, ideokinetic parietal apraxia, ideational apraxia.*

Agraphia

A separate discussion of the terms in agraphia is necessary purely for coordination of the elements. This is because agraphia may be due to an irreminiscence, to ideokinetic apraxia, to cortical motor pattern apraxia, or to lesions of engrams which the individual in question has, by training, intimately associated with the function of writing. We have

irreminiscence angular agraphia, ideokinetic apractic parieto-occipital agraphia, ideokinetic callosal apractic agraphia, formulation agraphia, third frontal apractic agraphia, second frontal apractic agraphia, and *external capsular agraphia* (the fibers conveying impulses from the angular gyrus to the frontal lobe for writing coursing in the external capsule). It will be evident from this that the subject of agraphia is the ultimate in difficulty of cerebral localization on the basis of it alone. Isolated agraphia occasionally results from a lesion of the foot of the second frontal convolution and from a lesion of the gyri of Gratiolet (border between the angular gyrus and the occipital lobe). *Isolated agraphia,* however, is exceedingly rare.

Alexia

Alexia (a type of aphasia in the broad sense) must also be the subject of correlation because it may result from lesions in any one of a great variety of locations and may be agnostic or semantic and may result from loss of motor patterns which in a given case have been associated with the patient's system of reading. Alexia may affect letters, words, musical symbols, and numerals. We have *occipital agnostic verbal alexia, angular agnostic verbal alexia, angular agnostic musical alexia, angular agnostic literal alexia, parietal agnostic numeral alexia, temporal agnostic verbal alexia, temporal semantic alexia, agnostic external capsular alexia, semantic external capsular alexia, agnostic third frontal alexia, semantic third frontal alexia,* and *tactile parietal agnostic alexia.*

It will be clear from this presentation that one must analyze with great care the reason for alexia in a given case. The causative lesion may be located anywhere from the occipital to the frontal lobe and it may be due to loss of recognition or loss of knowledge of significance of the recognized written matter.

Acalculia

Acalculia, the loss of ability to calculate, is also aphasia since it is a loss of language function. It results from a lesion of the parieto-occipital area.

Amimia

Amimia has been used as a descriptive term to designate inability of a patient to mimic the movements and gestures of the examiner. Amimia depends on a loss of either comprehension of the act (based on agnosia or on a higher mental function akin to "simultanagnosia") or an apraxia. While the term amimia may be a useful shorthand term for such a loss, it in no way is conclusive of the site of the causative lesion. The reason for the amimia must be analyzed.

Amusia

Amusia has been extensively used to indicate loss of the musical functions. It may be used as a convenient designation but its causative elements should be appreciated. It is based on agnosia for the symbols, aphasia for their comprehension, or apraxia for the execution of vocal or instrumental music.

PROPOSED NOMENCLATURE FOR AGNOSIAS, APRAXIAS AND APHASIAS

Localization of Lesions

Definitions and Old Terminology

1. Acalculia.

   Loss of ability to calculate with or without agnosia or loss of revisualization of symbols.

   O.T. Acalculia.

   Focus. The lesion affects the posterior parietal or parieto-occipital region.

2. Agnosia, Auditory temporal (general).

   Loss of power to recognize sounds heard.

   O.T. Auditory or acoustic agnosia, psychic deafness, Wernicke's aphasia.

   Focus. The lesion affects the posterior portion of the superior temporal convolution.

3. Agnosia, auditory, temporal, musical.

   Loss of power to recognize music as music by sound alone.

   O.T. Musical agnosia, psychic amusia.

   Focus. The lesion affects the anterior extremity of the temporal lobe.

4. Agnosia, auditory, temporal, verbal.

   Loss of power to recognize words heard.

   O.T. Auditory or acoustic verbal agnosia, psychic deafness, Wernicke's aphasia, subcortical acoustic verbal agnosia.

   Focus. The lesion affects the posterior superior temporal convolution.

5. Agnosia, tactile, occipital.

   Loss of power to recognize objects by the sense of touch alone even with the use of both hands. The loss is based on loss of revisualization (loss of visual reminiscence) and because of this loss the patient cannot form a visual image of the object by touch alone. The condition occurs in those particularly visual-minded.

   O.T. Apperceptive blindness of the senile of Pick, ideational agnosia of Liepmann.

   Focus. The lesion affects area 19 of Brodmann in its convex portion.

6. Agnosia, tactile, parietal.

   Loss of power to recognize by touch alone objects which are recognized by the other senses, or in the other hand, as this agnosia is unilateral.

   O.T. Astereognosis, tactile agnosia.

   Focus. The lesion affects the parietal lobe and may be cortical or subcortical.

7. Agnosia, tactile, parieto-occipital.

Definition is identical with that of occipital tactile agnosia (5).

O.T. Apperceptive blindness of the senile of Pick, ideational agnosia of Liepmann.

Focus. The lesion affects area 19 of Brodmann and the posterior portion of the parietal lobe.

8. Agnosia, tactile, parieto-occipital, finger.

Loss of power to recognize one's fingers by the sense of touch alone.

O.T. Finger agnosia.

Focus. The lesion affects the convolutions of Gratiolet.

9. Agnosia, visual, angular, literal.

Loss of power to recognize letters by vision alone, due to a lesion of the angular gyrus.

O.T. Visual literal agnosia, literal alexia, part of Wernicke's aphasia.

Focus. The lesion affects the angular gyrus.

10. Agnosia, visual, angular, verbal.

Loss of power to recognize written or printed words by vision alone (embossed letters may still be read) due to a lesion of the angular gyrus.

O.T. Visual verbal agnosia, alexia, part of Wernicke's aphasia.

Focus. The lesion affects the angular gyrus.

11. Agnosia, visual, angular, musical.

Loss of power to recognize musical notes by vision alone, due to a lesion of the angular gyrus.

O.T. Visual agnosia for musical symbols, visual amusia.

Focus. The lesion affects the angular gyrus.

12. Agnosia, visual, frontal, literal.

Loss of power to recognize letters by sight alone, due to a lesion of the frontal lobe. Through destruction of Broca's convolution or of the frontal writing center at the foot of the second frontal convolution the motor associations (which the patient has maintained since childhood and which to him are necessary for visual recognition) even visual recognition has become impossible. This is a rare occurrence.

O.T. Visual literal agnosia, literal alexia, part of Wernicke's aphasia.

Focus. The lesion affects the foot of the second or third frontal convolutions. The explanation is identical with not in the angular gyrus is determined by the presence in the case of apractic aphasia or other apraxia, especially cortical motor pattern apraxia.

13. Agnosia, visual, frontal, verbal.

Definition is identical with (12) above except that words alone and not letters are effected.

O.T. Visual verbal agnosia, element of Wernicke's aphasia, alexia.

Focus. The lesion affects the foot of the second or third frontal convolutions. The explanation is identical with that given in (12) above. The reason that recognition of letters is preserved is that they are stored as engrams of objects in area 18 of Brodmann in many instances.

14. Agnosia, visual, insular, literal.

Loss of power to recognize letters by sight alone due to a lesion of the insula or pathways immediately subcortical to it. By destruction of tracts, which in such a case are necessary for association of the convolution of Broca or of the foot of the second frontal convolution with the angular gyrus, the angular gyrus cannot function alone in recognition of letters. This is a rare occurrence and is based on retained childhood associations.

O.T. Visual literal agnosia, alexia, part of Wernicke's aphasia.

Focus. The lesion affects the insula and probably of necessity the external capsule through which the association fibers go.

15. Agnosia, visual, insular, verbal.

The definition is identical with that of (14) above except that the loss affects words and not letters.

O.T. Visual verbal agnosia, alexia, part of Wernicke's aphasia.

Focus. The explanation of the syndrome and its rarity are identical with those given under (14) above.

16. Agnosia, visual, occipital (general). This includes visual agnosia for animate and inanimate objects.

Loss of power to recognize objects or symbols by vision alone in spite of adequate visual perception (and with sufficient mental capacity in general) due to a lesion of the occipital lobe. The visual agnosias for symbols are called agnosias rather than aphasias because the term agnosia is more specific.

O.T. Visual agnosia (qualified), psychic blindness, mind-blindness.

Focus. The lesion affects area 18 of Brodmann in its convex portion.

17. Agnosia, visual, occipital, musical.

Loss of power to recognize musical symbols by vision alone due to a lesion of the occipital lobe. There is nearly always, if not always, an associated homonymous hemianopia.

O.T. Visual musical agnosia, subcortical musical alexia.

Focus. The focus is subcortical (and perhaps also cortical) in the occipital lobe. The agnosia is caused by the

fact that impulses from both areae striatae fail to reach the angular gyrus for recognition.

18. Agnosia, visual, occipital, literal.

The definition is identical with that given in (17) above except that letters instead of musical notes are affected.

O.T. Literal agnosia, subcortical literal agnosia, literal alexia.

Focus. The focus and explanation are identical with that given in (17) above.

19. Agnosia, visual, occipital, numeral.

Loss of power to recognize mathematical symbols due to a lesion of the occipital lobe. There is usually an associated homonymous hemianopia.

O.T. Visual numeral agnosia, numeral alexia, psychic blindness.

Focus. The focus is in the occiptal lobe so placed as to prevent impulses from reaching the borders of the interparietal sulcus from both areae striatae.

20. Agnosia, visual, occipital, verbal.

The definition is identical with that given under (17) above except that words instead of musical notes are affected.

O.T. Pure visual verbal agnosia, pure word blindness.

Focus. The lesion is in the occipital lobe, subcortical (and often cortical). It prevents recognition of words by interrupting impulses from both areae striatae to the angular gyrus. It does not cause agraphia because the angular gyrus itself is spared. That is the basis for the old terminology "pure."

21. Agnosia, visual, parietal, numeral.

Loss of power to recognize mathematical symbols due to a lesion of the parietal lobe, either at the borders of the interparietal sulcus or subcortical to that area.

O.T. Visual agnosia for mathematical figures, figure agnosia.

Focus. The lesion is cortical on the borders of the interparietal sulcus or subcortical to that area.

22. Agnosia, visual, parieto-occipital, finger.

Loss of power to recognize fingers by vision alone, those of the patient or those of the examiner or both.

O.T. Finger agnosia.

Focus. The lesion affects the convolutions of Gratiolet.

23. Agnosia, visual, subcortical, angular, musical.

Loss of power to recognize musical symbols due to a subcortical lesion of the angular gyrus. There is usually an associated homonymous hemianopia.

O.T. Musical agnosia, visual agnosia for musical notes.

Focus. The lesion is subcortical to the angular gyrus and prevents recognition by interrupting impulses from both

areae striatae to the angular gyrus. There is no agraphia for musical notes because the cortex of the angular gyrus is not affected.

24. Agnosia, visual, subcortical, angular, verbal.
    The definition is identical with that of (23) above except that words instead of musical notes are affected.
    O.T. Pure visual verbal agnosia, alexia, subcortical visual verbal agnosia.
    Focus. The lesion affects the subcortical region of the angular gyrus. There is no agraphia because the cortex is spared.

25. Agnosia, visual, temporal, literal (and verbal).
    Loss of power to recognize letters and words due to a lesion of Wernicke's area or the small space between Wernicke's area and the angular gyrus.
    O.T. Visual literal (and verbal) agnosia, literal (and verbal) alexia, Wernicke's aphasia.
    Focus. The lesion either separates Wernicke's area in the posterior superior temporal convolution from the angular gyrus or affects Wernicke's area itself. It causes visual agnosia because in the great majority of cases the angular gyrus cannot function for recognition of symbols without association with Wernicke's area. This is because visual engrams are laid down in close association with auditory ones.

26. Agraphia.
    A descriptive term when not qualified, meaning loss of the power to write.
    O.T. Agraphia.
    Focus. On the basis of agraphia alone it is usually impossible to make a focal diagnosis. See agraphia, isolated.

27. Agraphia, apractic, second frontal.
    Loss of power to write due to a lesion of the writing center at the foot of the second frontal convolution. The patient usually is unable to write by any method, even by building words with blocks or by writing on a typewriter.
    O.T. Motor agraphia, isolated agraphia.
    Focus. The lesion is in the cortex of the foot of the second frontal convolution.

28. Agraphia, apractic, third frontal.
    Loss of the power to write due to a lesion of Broca's convolution. There is an associated apractic aphasia.
    O.T. Broca's aphasia, motor agraphia.
    Focus. Broca's convolution is affected either cortically or subcortically. The agraphia results secondarily because most patients are unable to write unless they have the motor patterns for speech associated with their writing mechanism.

29. Agraphia, external capsular.
    Loss of the capacity to write due to a lesion of the external capsule.
        O.T. Motor agraphia.
            Focus. The lesion affects the external capsule and causes agraphia because the impulses from the angular gyrus (where reminiscence of words occurs) fail to reach the writing center at the foot of the second frontal convolution.

30. Agraphia, formulation, temporal.
    Loss of ability to write because of inability to formulate language, all due to a lesion of the temporal lobe.
        O.T. Amnesic agraphia, paragraphia.
            Focus. The lesion affects the posterior part of the temporal lobe. It may be small or large. It interferes with the fuction of the language formulation area.

31. Agraphia, ideokinetic apractic, callosal.
    Loss of ability to write because of a lesion of the corpus callosum which causes ideokinetic apraxia.
        O.T. Apractic agraphia.
            Focus. The lesion is in the corpus callosum, usually in the anterior portion. Unless the lesion is a slowly progressive one the patient usually recovers ability to write.

32. Agraphia, ideokinetic apractic, parieto-occipital.
        O.T. Apractic agraphia.
            Focus. The lesion affects most often the subcortical structures of the general region of the supramarginal gyrus.

33. Agraphia, irreminiscence literal (or verbal) angular.
    Loss of ability to write due to loss of ability to recall the appearance of letters or words, and caused by a lesion of the angular gyrus.
        O.T. Agraphia.
            Focus. The lesion affects the cortex of the angular gyrus.

34. Agraphia, isolated, parieto-occipital.
    Loss of ability to write due to loss of knowledge of visual or visual memory guidance of hand movements and due to a lesion of the convolutions of Gratiolet.
        O.T. Isolated agraphia (it being the only aphasic element in the case) as part of Gerstmann's syndrome.
            Focus. The lesion affects the convolutions of Gratiolet at the border between the angular gyrus and the occipital convolutions.

35. Agraphia, isolated, second frontal, apractic.
    Loss of ability to write due to cortical motor apraxia for writing only and due to a lesion at the foot of the second frontal convolution.
        O.T. Motor agraphia, isolated agraphia.
            Focus. The lesion affects the cortex at the foot of the second frontal convolution.

36. Agraphia, temporal.

Loss of ability to write due to crippling of the angular gyrus through destruction of its associative connection with Wernicke's area and caused by a lesion of the superior temporal convolution between the angular gyrus and Wernicke's area.

O.T. Agraphia accompanying Wernicke's aphasia.

Focus. The lesion affects the small area of cortex between the area of Wernicke and the angular gyrus (part of area 22 of Brodmann).

37. Alexia.

Descriptive term signifying loss of ability to read without reference to the physiologic cause except that it is due to a focal cerebral lesion. Sufficient visual perception and mental capacity are presupposed.

O.T. Alexia.

38. Alexia, agnostic, external capsular, literal and verbal.

Loss of ability to recognize words and letters due to a lesion of the external capsule and based physiologically on dissociation of the angular gyrus from Broca's convolution. This is a rare occurrence and is found only in persons who have retained childhood associations between vocal reading and visual reading.

O.T. Alexia, visual, verbal or literal alexia.

Focus. The lesion affects the external capsule.

39. Alexia, agnostic, literal and verbal angular.

Loss of ability to recognize letters and words due to a lesion of the angular gyrus.

O.T. Agnostic alexia, alexia, visual literal and verbal agnosia.

Focus. The lesion affects the cortex of the angular gyrus.

40. Alexia, agnostic, musical, angular.

Definition and physiologic-anatomic basis is identical with (39) above except that musical symbols are affected instead of letters and words.

O.T. Musical alexia, visual musical agnosia.

Focus. The lesion affects the cortex of the angular gyrus.

41. Alexia, agnostic, numeral, parietal.

Loss of ability to recognize mathematical figures due to a lesion of the parietal lobe (borders of the interparietal sulcus).

O.T. Visual agnosia for mathematical figures, mathematical alexia.

Focus. The lesion affects the cortex of the parietal lobe at the borders of the interparietal sulcus.

42. Alexia, agnostic, third frontal, literal and verbal.

Loss of ability to recognize letters and words due to a lesion of Broca's convolution. This is a rare occurrence and is found only in those who have retained childhood associations between the angular gyrus and Broca's convolution as a necessity for recognition.

O.T. Visual verbal agnosia, alexia.

Focus. The lesion affects the cortex of Broca's convolution.

43. Alexia, agnostic, verbal, occipital.

Loss of ability to recognize words due to a lesion of the occipital lobe so placed that impulses from both areae striatae fail to reach the angular gyrus.

O.T. Pure visual verbal agnosia.

Focus. The lesion is in the occipital lobe and if unilateral affects also the splenium of the corpus callosum. There is nearly always an associated homonymous hemianopia.

44. Alexia, agnostic, verbal, temporal.

Loss of ability to recognize written or printed words due to a lesion of the temporal lobe. While the angular gyrus functions in recognition, in most cases it cannot function without association with Wernicke's area.

O.T. Visual verbal agnosia, verbal alexia. Part of Wernicke's aphasia.

Focus. The lesion affects the cortex of Wernicke's area itself or separates Wernicke's area from the angular gyrus.

45. Alexia, semantic, external capsular.

Loss of ability to determine significance of words, which are recognized, due to a lesion of the external capsule. In rare cases a lesion in this location even prevents recognition but usually interferes, if at all, with reading, only in determination of significance.

O.T. Semantic alexia, transcortical sensory aphasia.

Focus. The lesion affects the external capsule, usually also the insula.

46. Alexia, semantic, temporal.

Loss of ability to determine significance of words, which are recognized, due to a lesion of the temporal lobe. The physiologic basis is the requirement of the angular gyrus, in most cases, to have association with Wernicke's area in order to determine significance of what is read.

O.T. Semantic alexia, transcortical sensory aphasia.

Focus. The lesion affects the cortex of Wernicke's area or separates Wernicke's area from the angular gyrus.

47. Alexia, semantic, third frontal.

Loss of ability to determine the significance of words, which are recognized, due to a lesion of Broca's convolution. This form of alexia in a minor degree is common.

O.T. Semantic alexia, transcortical sensory aphasia.

Focus. The lesion affects Broca's convolution either cortically or subcortically.

48. Alexia, tactile, agnostic, parietal.

Loss of ability to read letters or words by touch, sufficient tactile perception presupposed, due to a lesion of the parietal lobes, cortical or subcortical.

O.T. Tactile aphasia, tactile agnosia for letters, astereognosis.

Focus. The lesion affects the parietal lobe opposite to the affected hand. It may be cortical or subcortical.

49. Amimia.

A purely descriptive term used to designate loss of ability to mimic gestures. As amimia may be based on agnosia or on apraxia, the cause must be analyzed in each instance.

O.T. Amimia.

50. Amusia.

A purely descriptive term used to designate a disturbance of the musical sense, auditory, visual, or motor. Its subdivisions are classified as agnosias, apraxias, and aphasias.

O.T. Amusia.

51. Aphasia.

A term restricted to mean "loss of language association with." It includes agnosias and apraxias which are concerned with language. Thus "finger aphasia" means loss of the ability to name or comprehend the names of the fingers. Visual object aphasia means loss of ability to name objects seen, etc. Aphasias based on apraxia are apractic aphasias. The term is therefore narrower than the old expansive definition.

52. Aphasia, apractic, insular.

Loss of ability to speak based on apraxia and due to a lesion of the insula (probably chiefly the subcortical structures, the external capsule in particular).

O.T. Subcortical or association motor aphasia, anarthria of Pierre Marie.

Focus. The lesion affects the insula probably always subcortically.

53. Aphasia, apractic, frontal.

Loss of ability to speak due to a lesion of Broca's convolution cortical or subcortical.

O.T. Broca's aphasia, subcortical motor aphasia, transcortical motor aphasia.

Focus. The lesion affects Broca's convolution, cortically or subcortically.

54. Aphasia, apractic, musical, triangularis.

Loss of ablity to sing words due to a lesion of the pars triangularis of the third frontal convolution.

O.T. Avocalia, motor amusia.

Focus. The lesion affects the pars triangularis of the third frontal convolution.

55. Aphasia, auditory, musical, semantic, temporal.

Loss of ability to comprehend played or sung music, which is recognized, due to a lesion of the temporal lobe. This is rare because of the facility with which the minor side assumes the function.

O.T. Auditory amusia.

Focus. The lesion affects the anterior portion of the temporal lobe.

56. Aphasia, auditory, object, temporal.

Loss of ability to associate the name of an audible object with the sound of the object, e.g. the patient cannot say "bell" when he hears the ring or "watch" when he hears the tick.

O.T. Amnesia aphasia.

Focus. The lesion affects the posterior part of the temporal lobe.

57. Aphasia, auditory, semantic, temporal.

Loss of ability to comprehend names of sounds in general in spite of recognition of the sounds, the disability being due to a temporal lesion.

O.T. Amnesic aphasia.

Focus. The lesion affects the temporal lobe.

58. Aphasia, color, occipital.

Loss of ability to name colors which are seen and recognized, due to an occipital lesion. The lesion breaks the association between the occipital area for recognition and the language formulation area.

O.T. Amnesic color blindness, optic aphasia for colors, visual aphasia for colors.

Focus. The lesion affects the occipital lobe close to the temporal lobe.

59. Aphasia, color, temporal.

This is identical with (58) above except that the lesion is in the temporal lobe close to the occipital.

60. Aphasia, formulation, temporal.

Loss of ability to formulate words and phrases into language.

O.T. Amnesic aphasia, paraphasia, agrammatism.

Focus. The lesion affects the posterior portion of the temporal lobe, chiefly area 37 and the posterior extremity of area 22 of Brodmann.

61. Aphasia, visual, semantic, external capsular.

Loss of comprehension of significance of written or printed words due to a lesion of the external capsule. This form of aphasia is based on the necessity of having association between the angular gyrus and Broca's convolution for full comprehension of written or printed words.

O.T. Semantic aphasia, semantic alexia.

Focus. The lesion affects the external capsule.

62. Aphasia, visual, semantic, temporal.

Loss of comprehension of significance of written or printed words due to a lesion of the temporal lobe. This is based on the dependence of the angular gyrus in association with Wernicke's area for comprehension of written language.

O.T. Transcortical sensory aphasia, semantic alexia, part of Wernicke's aphasia, Wernicke's aphasia.

Focus. The lesion affects Wernicke's area or the short portion of cortex between it and the angular gyrus (posterior extremity of area 22 of Brodmann).

63. Aphasia, visual, semantic, third frontal.

Loss of ability to comprehend significance of written or printed matter due to a lesion of Broca's convolution. This is based on the physiologic necessity for association of the angular gyrus with Broca's convolution for full comprehension of written language.

O.T. Semantic aphasia, semantic alexia.

Focus. The lesion affects Broca's convolution.

64. Aphasia, tactile, finger, parietal.

Loss of ability to name or comprehend names of fingers held by the examiner due to a lesion of the parietal lobe.

O.T. Amnesic finger aphasia, finger aphasia.

Focus. The lesion affects the parietal lobe usually in the region of the supramarginal gyrus. It interrupts the association between the parietal lobe and Wernicke's area.

65. Aphasia, visual, finger, temporo-occipital.

Loss of ability to name or comprehend the names of fingers, seen by the patient, due to a lesion of the temporo-occipital area.

O.T. Finger aphasia, visual finger aphasia, visual agnostic element of finger agnosia, amnesic aphasia for fingers.

Focus. The lesion affects the temporo-occipital area and interrupts association between the occipital area for recognition (area 18) and Wernicke's area.

66. Aphasia, visual, temporal, object.

Loss of ability to name objects seen, though they are recognized, due to a lesion of the temporal lobe.

O.T. Amnesic aphasia, anomia.

Focus. The lesion affects area 37 of Brodmann or the posterior extremity of area 22, in general the posterior portion of the temporal lobe. It interrupts communication between area 18 and Wernicke's area for which reason the auditory memories of the names cannot be associated with the memory of the object seen.

67. Apraxia (for any particular movement, act, idea, or apraxia in general).

The term is only descriptive until qualified. Apraxia is loss of ability to perform as desired or as requested, through loss of

memory of how to perform. This loss of memory in turn is due to destruction of cerebral engrams. Ataxia, paralysis, and dementia must always be excluded as causes.

68. Apraxia, cortical motor pattern.

Loss of ability to execute a *movement* due to lesion of the cortical motor pattern. This type of apraxia is illustrated by Broca's aphasia, by agraphia as a result of a lesion in the foot of the second frontal convolution, by aphasia from a lesion of the precentral gyrus at the frontal operculum, or by apraxia of singing from a lesion of the pars triangularis of the third frontal convolution. The result is the same whether the cortical motor pattern itself is destroyed or whether its connections with the Betz cells, through which the projection produce the movements, are broken.

O.T. Kinetic apraxia of the limb, apraxia of lateral gaze, Broca's aphasia, subcortical motor aphasia, transcortical motor aphasia, apractic amusia, apractic agraphia, apractic aphasia, avocalia.

Focus. The lesion for each type is cortical if it destroys the motor pattern or subcortical if it prevents the organized plan of the movement from reaching the Betz cells where the projection fibers begin for execution of the movement.

69. Apraxia, ideational.

Loss of ability to carry out an idea due to a diffuse lesion of the brain affecting the ideational plan of action. The individual acts constituting the ideational plan can be executed without trouble but the entire ideational plan cannot be retained in consciousness long enough to be executed.

O.T. Ideational apraxia.

Focus. There is no single focus. The "lesion" is either a toxemia or is diffusely placed such as in multiple vascular softening, or multiple tumors.

70. Apraxia, ideokinetic, callosal.

Loss of ability to execute the plan of an act, the plan and the mechanism of execution both being undisturbed but the connection between them being broken, by a lesion of the corpus callosum. The loss may affect the fingers of the patient only.

O.T. Ideokinetic apraxia. Apraxia of finger demonstration, finger agnosia.

Focus. The lesion is in the corpus callosum, anterior for the face and upper limbs, posterior for the lower limbs, intermediate for the trunk. The apraxia does not remain unless the lesion is progressive.

71. Apraxia, ideokinetic, parietal.

This condition is identical with (70) above except that the lesion affects the subcortical structures of the parietal lobe, most potently at the supramarginal gyrus. When it affects the minor limb it is the old sympathetic apraxia.

72. Autotopagnosia, visual, occipital.

Loss of ability to recognize parts of the body. It is essentially visual agnosia for the body and hence is part of general visual agnosia.

73. Irreminiscence.

A term coined to designate a disturbance of ability to recall, i.e. a disturbance of power of reminiscence. It is only descriptive unless qualified.

O.T. Revisualization, re-auditorization, loss of power to recall, amnesia of certain types.

74. Irrimeniscence, auditory, musical, temporal.

Loss of power to recall into auditory memory musical compositions formerly known, due to a lesion of the temporal lobe.

O.T. Loss of musical recall.

Focus. The lesion affects the anterior end of the temporal lobe. (Irritation of the same area causes auditory musical hallucinations).

75. Irreminiscence, auditory (general) temporal.

Loss of power to recall auditory memories in general due to a lesion of the temporal lobe.

O.T. Loss of reauditorization, loss of recall of auditory memories.

Focus. The lesion is a large one in the temporal lobe.

76. Irreminiscence, auditory, verbal, temporal.

Loss of power to recall the auditory memory of words due to a lesion of the temporal lobe. (Irritation of this area causes hallucinations of words.)

O.T. Amnesia aphasia, loss of recall of auditory memory of words.

Focus. The lesion affects the temporal lobe, chiefly area 37 of Brodmann. It is the underlying factor in some cases of disturbance of language formulation.

77. Irreminiscence, color, occipital.

Loss of ability to recall visual images of colors. In some instances the patients recall all colors as being black. (Irritation of this area causes hallucinations of colors.)

O.T. Loss of revisualization of colors.

Focus. The lesion affects the occipital lobe, area 19 of Brodmann, especially the lateral convex surface, cortical or subcortical.

78. Irreminiscence, direction, occipital.

Loss of ability to recall directions, either the cardinal ones or as related to the body of the individual, due to a lesion of the occipital lobe.

O.T. Geometric-optic agnosia, disorientation in space.

Focus. The lesion affects the occipital lobe, chiefly area 19 of Brodmann on the inferior surface.

79. Irreminiscence, distance, occipital.
This condition is identical with (78) above except that distance instead of direction, is affected.

80. Irreminiscence, visual, body, parieto-occipital.
Loss of ability to revisualize the body of the patient or of another, due to a lesion of the parieto-occipital lobe. (Irritation of this area causes visual hallucinations of living things.)
O.T. Disturbance of the body scheme, autotopagnosia.
Focus. The lesion affects the parieto-occipital region, either cortically or subcortically, usually within a centimeter of the mesial surface (area 19 of Brodmann).

81. Irreminiscence, visual, finger, parieto-occipital.
Loss of ability to recall visually one's own fingers or those of another due to a lesion of the parieto-occipital area.
O.T. Finger agnosia, visual agnostic element of finger agnosia, disturbance of the body scheme, part of the Gerstmann syndrome.
Focus. The lesion affects the border between the angular gyrus and the occipital lobe, the convolutions of Gratiolet.

82. Irreminiscence, visual, object, occipital.
Loss of power to recall visual images of objects due to an occipital lesion (irritation of this area causes visual hallucinations of objects).
O.T. Loss of revisualization of objects, disorientation in space, acoustic-optic agnosia.
Focus. The lesion affects area 19 of Brodmann, chiefly the inferior convex portion, cortically or subcortically.

83. Irreminiscence, visual, literal, angular.
Loss of ability to recall visual images of letters due to a lesion of the angular gyrus (cortex). This loss causes agraphia.
O.T. Loss of revisualization of letters.
Focus. The lesion affects the cortex of the angular gyrus.

84. Irreminiscence, visual, musical, angular.
Loss of power to recall appearance of musical notes or musical composition due to a lesion of the angular gyrus.
O.T. Loss of revisualization of musical symbols.
Focus. The lesion affects the angular gyrus.

85. Irreminiscence, visual, numeral, parietal.
Loss of ability to recall visual images of mathematical figures due to a lesion of the borders of the interparietal sulcus.
O.T. Loss of revisualization of mathematical figures.
Focus. The lesion affects the borders of the interparietal sulcus.

86. Irreminiscence, visual, verbal, angular.
Loss of power to revisualize written or printed words, due to a lesion of the angular gyrus. This causes agraphia.
O.T. Loss of revisualization of written or printed words, sensory agraphia.
Focus. The lesion affects the cortex of the angular gyrus.

87. Irreminiscence, visual, verbal, temporal.

Loss of ability to revisualize words, due to a lesion of the temporal lobe. This causes agraphia.

O.T. Loss of revisualization of written or printed words, sensory agraphia.

Focus. The lesion affects the portion of cortex between Wernicke's area and the angular gyrus (posterior extremity of area 22 of Brodmann). The syndrome is based physiologically on the dependence of the angular gyrus on the area of Wernicke in the function of revisualization of words.

Classic Nomenclature with Proposed Nomenclature Which is Equivalent, is Embraced By, or Embraces the Classic

(O.T., Old Terminology. P.N., Proposed Nomenclature)

O.T. Acalculia.

P.N. Acalculia.

O.T. Agnosia. The term according to some authors included loss of recognition through several sense organs simultaneously.

P.N. Agnosia. The term is now limited to loss of recognition through one sense organ only.

O.T. Agnosia, acoustic or auditory.

P.N. Agnosia, auditory, temporal, (general).

O.T. Agnosia, acoustic or auditory, musical.

P.N. Agnosia, auditory, temporal, musical.

O.T. Agnosia, acoustic or auditory, verbal.

P.N. Agnosia, auditory, temporal, verbal.

O.T. Agnosia, disjunctive (agnosia, ideational).

P.N. In the new light this is interpreted as a combination of visual and tactile agnosia in various combinations.

O.T. Agnosia, dissolutive (agnosia, ideational).

P.N. This is somewhat similar to disjunctive agnosia and is interpreted as a combination of agnosias.

O.T. Agnosia, finger, of Gerstmann.

P.N. This is subdivided into visual agnosia for fingers, tactile agnosia for fingers, and ideokinetic apraxia of fingers.

O.T. Agnosia, geometric-optic.

P.N. This is a part of visual agnosia for objects, geometrical relations being part of objects small or large. That which formerly was considered geometric-optic agnosia is also in part visual object irreminiscence.

O.T. Agnosia, gustatory.

P.N. Gustatory agnosia whenever this may become established as a recognizable fact.

O.T. Agnosia, ideational.

P.N. Various combinations of visual plus tactile agnosia.

O.T. Agnosia, olfactory.
P.N. Olfactory agnosia whenever this may become established as a recognizable fact.

O.T. Agnosia, subcortical acoustic or auditory verbal.
P.N. Auditory verbal agnosia in which there happens to be good function of the minor cerebral hemisphere for formulation of language and for production of speech.

O.T. Agnosia, subcortical, visual, verbal.
P.N. Agnosia, visual, subcortical, angular, verbal.

O.T. Agnosia, tactile (astereognosis).
P.N. Agnosia, tactile, parietal.

O.T. Agnosia, visual (or optic).
P.N. Agnosia, visual, occipital (general).

O.T. Agnosia, visual for letters.
P.N. Agnosia, visual, occipital literal; or agnosia, visual subcortical or cortical angular literal; agnosia visual temporal literal, etc.

O.T. Agnosia, visual for mathematical figures.
P.N. Agnosia visual parietal, or parieto-occipital numeral.

O.T. Agnosia, visual, for musical notes.
P.N. Agnosia, visual occipital (or angular, or subcortical angular) musical.

O.T. Agnosia, visual, for objects or pictures.
P.N. Agnosia, visual occipital (general).

O.T. Agnosia, visual, verbal.
P.N. Agnosia, visual, angular, verbal.

O.T. Agrammatism.
P.N. Aphasia, formulation, temporal.

O.T. Agraphia.
P.N. Agraphia analyzed as to cause and site of lesion. See each heading beginning with "Agraphia."

O.T. Alexia.
P.N. Alexia analyzed as to cause and site. See each heading beginning with "Alexia."

O.T. Amimia.
P.N. Amimia as a descriptive term, but it has little significance until analyzed into terms of agnosia and apraxia.

O.T. Amusia.
P.N. Amusia. The term is to be analyzed in each case as to agnostic, apractic, or aphasic basis. Otherwise, it is purely descriptive.

O.T. Anomia.
P.N. Aphasia, visual, temporal, object.

O.T. Aphasia.
P.N. Aphasia. However, the term is restricted and redefined to mean "the language component of."

O.T. Aphasia, acoustic-optic.
P.N. Irreminiscence, visual, object, occipital.

O.T. Aphasia, amnesic.
P.N. Aphasia, visual, temporal, object; or Aphasia, auditory, temporal, object; or Aphasia, formulation.

O.T. Aphasia, Broca's.
P.N. Aphasia, apractic, third frontal.

O.T. Aphasia, jargon.
P.N. Aphasia, formulation.

O.T. Aphasia, motor.
P.N. Aphasia, apractic (with various anatomical locations of the causative lesions).

O.T. Aphasia, optic.
P.N. Aphasia, visual, temporal, object.

O.T. Aphasia, optic for colors.
P.N. Aphasia, color (temporal or occipital).

O.T. Aphasia, semantic.
P.N. Aphasia, semantic (qualified as to type and anatomic site of causative lesion).

O.T. Aphasia, sensory.
P.N. The term is too loose to have much significance and should be analyzed into its specific components.

O.T. Aphasia, subcortical motor.
P.N. Aphasia, apractic (qualified as to anatomical site of causative lesion).

O.T. Aphasia, transcortical motor.
P.N. Aphasia, apractic (qualified as to site of anatomical site of causative lesion). Its occurrence is based on the appearance of cases in which the minor hemisphere has excellent repetitive capacity.

O.T. Aphasia, Wernicke's.
P.N. As this is a complex syndrome dependent on agnosia, irreminiscence and aphasia the elements should be analyzed in each case.

O.T. Apraxia.
P.N. Apraxia. The defect should be analyzed in each case as to movement, act, and idea.

O.T. Apraxia, constructive (of Kleist).
P.N. This is based on visual irreminiscence of finger movements.

O.T. Apraxia, ideational.
P.N. Ideational apraxia.

O.T. Apraxia, ideokinetic.
P.N. Ideokinetic apraxia.

O.T. Apraxia, kinetic of limb.
P.N. Cortical motor pattern apraxia.

O.T. Apraxia, sympathetic.
P.N. Ideokinetic apraxia affecting the minor limb.

O.T. Astereognosis.
P.N. Tactile agnosia.

O.T. Autotopagnosia.
P.N. Visual autotopagnosia.

O.T. Avocalia.
P.N. Aphasia, apractic musical, triangularis.

O.T. Blindness, amnesic color of Wilbrand.
P.N. Aphasia, visual, temporal, object.

O.T. Blindness, apperceptive of the senile.
P.N. Simultaneous visual and tactile agnosia.

O.T. Blindness, cerebral, color, acquired.
P.N. Agnosia, visual color, occipital.

O.T. Blindness, psychic.
P.N. Agnosia, visual, occipital (general).

O.T. Charcot-Wilbrand syndrome.
P.N. Agnosia and irreminiscence visual, occipital.

O.T. Deafness, psychic.
P.N. Auditory agnosia and auditory semantic aphasia.

O.T. Deafness, verbal.
P.N. Auditory verbal agnosia.

O.T. Disorientation, optical, in space.
P.N. Irreminiscence, object, occipital.

O.T. Imperception (Hughlings Jackson).
P.N. Agnosia, visual, object, occipital.

O.T. Paraphasia.
P.N. Aphasia, formulation, temporal.

O.T. Simultanagnosia (Wolpert).
P.N. A complex disturbance of synthesis of visual impressions.

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PART II

A STUDY OF THE RELATIONSHIP BETWEEN STUTTERING OCCURRENCE AND PHONETIC FACTORS IN ORAL READING*

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It was the purpose of this study to ascertain whether stuttering occurs more often in relation to some speech sounds than to others. The formulation of a significant ranking of difficulty of speech sounds associated with stuttering should be of value in furthering the study of psychological and physiological factors in stuttering occurrence.

In considering the phonetic factor in stuttering, the following specific questions arose:

1. Can a ranking of difficulty of sounds associated with stuttering be formulated?

2. Can such a ranking of difficulty be compared to that presented by Johnson and Brown (4) in their study of stuttering in relation to speech sounds?

3. Will the study of speech sounds related to stuttering occurrence show that the phonetic factor is pronounced in influencing the stuttering of individuals?

4. Will a phonetic ranking of difficulty show the influence of physical factors in sound production?

5. Will the data establish that stuttering occurs more in relation to consonants than to vowels?

6. Will the preponderance of stuttering occur on initial sounds?

Without scientific approach to the study of phonetic factors, the speech clinician has to accept the opinion of the individual stutterer on his relative difficulties on certain sounds. Some stutterers are very positive of the existence of phonetic difficulty, making such statements as, "I can never produce an M sound without blocking," or "An S always causes a spasm." One may believe that the physical method of producing a sound is related to stuttering occurrence or that for an individual, spasms are localized. The stutterer will then block predominantly on lip movements, or tongue-tip movements, on tongue-back movements, and so forth.

Johnson and Brown (4) in a previous study had been able to formulate a ranking of difficulty of speech sounds for stutterers. Their first study was based on the reading of contextual material; the second study by Brown (1), on non-contextual material. The second study again showed differences in difficulty on various speech sounds. In both

*A part of a study completed under the direction of Dr. Lee Edward Travis.

studies the ranking of difficulty for an individual case might differ widely from the group ranking. Also, for any single case the ranking of difficulty in the reading of contextual material might differ markedly from such a ranking in the reading of non-contextual material. In the Johnson-Brown study the general phonetic factor was not considered to be strong in influencing stuttering, nor did it operate independent of other factors. Also, it could not be stated whether the factor influencing stuttering was actually phonetic, physiological, or psychological. An attempt was made in the present study to establish a ranking of difficulty of sounds in stuttering to be compared to that of Johnson and Brown (4). However, the situation in the Johnson-Brown research remained constant; in this study four varied test situations were used. Also, in the earlier investigation each of the thirty-two stutterers read five passages of 1,000 words each; in this study each of the forty-three subjects read four 550-word selections.

The testing situations used in the present investigation were not created primarily for the study of phonetic factors in stuttering occurrence, but were designed to ascertain the effects of changes in social complexity upon stuttering frequency. However, in the writing of the reading material, care was taken that every consonant (with the exception of ZH and NG) should be included initially; also, every common consonant combination appeared initially. Since Johnson and Brown (4) established that stuttering occurs predominantly on consonants, little effort was made to arrange the context of the selections in order to include every vowel initially. As a matter of fact, little attention was given to the classification of initial vowels.

Johnson and Brown (4) were led to assume that some psychological factor or factors were operating so that certain sounds acted as cues to precipitate the stuttering occurrence. It seemed impossible that this psychological factor operated on a highly conscious or voluntary level. The fact that certain stutterers were relatively consistent in stuttering on certain sounds, pointed to a psychological factor operating on an involuntary level. The stutterers were then unaware of, or only vaguely aware of, the cues associated with stuttering.

Procedure

The data used in the author's study on the relationship between the severity of stuttering and the social complexity of the oral reading situations formed the bases of this study. Of the fifty-two cases tested, forty-three completed all four situations. Results from these forty-three cases were considered. In recording the spasms of the stutterers tested, the concealed observer drew a line through the sound connected with stuttering whenever a spasm occurred. The words thus marked were listed for each case.

Phonetic analysis of reading selections. A phonetic analysis of the test selections was prepared. In this way, the number of times each consonant occurred initially was ascertained. It also became apparent that the stutterers had stuttered on medial consonants in twenty-three

words. These words upon which stuttering occurred medially at least twice are listed:

important	remembered	before	insides
expensive	into	hopefully	also
apartment	today	conversation	always
disapprove	another	either	anyway
admission	suggested	another	conversation
watermelon	thirty-five	exciting	

In almost every instance (with the exception of secondary accent when the V was stuttered in "conversation" and the voiced TH in "another"), the medial consonants upon which stuttering occurred began accented syllables. This meant that for the individual, those medial consonants began the part of the word which necessitated increased volume and the initiation of muscular effort. Consequently, it was thought justifiable to add these medial consonants associated with stuttering to the list of initial consonants. Medial consonants not occurring with stuttering at least twice were not so listed. The number of initial vowels was also ascertained with no attempt made to classify them. With these figures thus assembled, a ranking of difficulty of sounds, according to mean and median percentage of stuttering occurrence, could be readily obtained.

Results and Discussion

It was possible within certain limitations to arrange the sounds according to a ranking of difficulty. Table I presents the arrangement of sounds according to median per cent of stuttering based on data from forty-three cases reading in four varied situations. Table II shows the arrangement of sounds according to mean per cent of difficulty. Because the number of cases is small and because the mean tends to be weighted by the difficulties of severe stutterers, the table presenting the median per cent gives the better measure. The five sounds which appear at the top of both tables are: G, D, TH (voiceless), L, and CH. These are not, however, in the same ranking of difficulty for both tables. The order of difficulty shown in Table I may be compared to that of Johnson and Brown (4), selecting only the consonants from their table.

COMPARISON OF THE FINDINGS OF THE JOHNSON-BROWN AND OF THE PRESENT STUDY IN REGARD TO RANKING OF DIFFICULTY OF CONSONANTS IN STUTTERING OCCURRENCE

Johnson-Brown	Present Study	Johnson-Brown	Present Study
Z	—	N	K
L	G	P	R
G	D	S	J
D	TH (θ)	B	F
J	L	WH	S
M	N	SH	W
R	CH	W	TH (δ)
TH (θ)	M	T	H
K	T	F	WH
CH	B	TH (δ)	SH
V	P	H	V

TABLE I

RANKING OF DIFFICULTY OF SOUNDS ACCORDING TO MEDIAN PER CENT OF STUTTERING OF FORTY-THREE STUTTERERS IN FOUR VARIED ORAL READING SITUATIONS

Consonants associated with stuttering	Situations			
	1	2	3	4
G	8.33	8.33	8.33	8.33
D	0	8.33	8.33	16.67
TH (θ)	0	9.09	9.09	9.09
L	0	7.68	7.68	7.68
N	0	6.67	6.67	6.67
CH	0	14.29	0.00	0.00
M	0	5.56	5.56	5.56
T	0	5.56	5.56	5.56
B	0	5.26	5.26	5.26
P	0	4.00	4.00	4.00
K	0	7.14	0.00	7.14
R	0	0.00	5.56	5.56
J	0	5.26	0.00	5.26
F	0	3.45	3.45	3.45
S	0	3.77	1.89	3.77
W	0	2.63	2.63	2.63
TH (δ)	0	0.00	1.85	3.70
H	0	0.00	1.89	0.00
WH	0	0.00	0.00	0.00
SH	0	0.00	0.00	0.00
V	0	0.00	0.00	0.00

TABLE II

RANKING OF DIFFICULTY OF SOUNDS ACCORDING TO MEAN PER CENT OF STUTTERING OF FORTY-THREE STUTTERERS IN FOUR VARIED READING SITUATIONS

Consonants associated with stuttering	Situations			
	1	2	3	4
CH	7.97	19.27	19.93	21.93
G	9.50	17.64	15.70	23.45
D	10.47	17.44	20.74	13.37
R	7.11	13.82	13.95	14.34
L	6.08	14.13	11.99	16.10
TH (θ)	5.29	11.21	10.99	15.22
N	4.81	11.78	9.77	15.78
J	5.63	11.26	10.89	14.20
WH	6.51	10.70	9.30	14.88
P	5.21	9.12	10.33	13.77
M	5.17	9.82	9.43	10.85
T	4.91	7.24	8.53	13.70
B	4.65	9.06	11.99	11.26
V	2.91	7.56	6.98	12.21
K	3.62	5.36	4.86	11.22
W	2.82	6.12	6.85	8.08
S	2.72	5.70	6.71	8.73
F	2.57	5.69	5.13	9.22
SH	2.95	8.88	4.23	5.71
TH (δ)	2.02	3.32	5.08	6.50
H	1.32	2.63	2.76	3.64

It will be seen that in both studies the G, D, L, TH (voiceless), CH, and M in the larger percentages and the S, F, SH, WH, TH (voiced), W, and H in the smaller percentages are fairly relative in position in the respective tables. It was unfortunate that the reading selections of the present study did not utilize the initial Z sound more than once. It could not be listed in the present consideration of sounds because its occurrence was too infrequent to furnish significant data.

In studying Tables I, and II, attention should be called to the effect of changes in reading situations upon the percentages. Table I, for the majority of the sounds listed, shows an increase in percentages from situation to situation comparable to the finding presented in a previous study (3). For all twenty-one sounds the percentage of stuttering increased markedly from Situations 1 to 2, from 1 to 3, and from 1 to 4; for eleven sounds the percentage of stuttering was greater in Situation 2 than in Situation 3; for ten sounds, greater in Situation 3 than in Situation 2; and for nineteen sounds the percentage was higher in Situation 4 than in Situation 3. The differences between situations shown in the ranking of difficulty of sounds according to mean per cent is comparable to the differences between mean percentages of stuttering in the four situations.

In Table III the percentage of cases stuttering on specific sounds is presented, with the percentage of the occurrence of each sound in the total number of 573 sounds per test considered. The order of the

TABLE III

PERCENTAGE OF OCCURRENCE OF EACH CONSONANT AND PERCENTAGE OF CASES STUTTERING ON EACH CONSONANT

Sound	Percentage of occurrence*	Situations			
		1	2	3	4
G	2.1	46.5	67.4	72.1	74.4
D	2.1	58.1	69.8	58.1	74.4
TH (θ)	1.9	32.6	58.1	51.1	65.1
L	2.3	34.9	58.1	58.1	58.1
N	2.6	23.3	53.4	53.4	65.1
CH	1.2	30.2	51.1	48.8	46.5
M	3.2	41.8	51.1	55.8	53.4
T	3.2	34.9	55.8	48.8	67.4
B	3.3	44.2	51.1	60.4	67.4
P	4.4	39.5	65.1	60.4	60.4
K	2.4	30.2	53.4	46.5	72.1
R	3.2	44.2	48.8	60.4	53.4
J	3.3	41.8	51.1	48.8	62.7
F	5.1	34.9	55.8	55.8	60.4
S	9.2	44.2	72.1	74.4	72.1
W	6.6	39.5	58.1	55.8	60.4
TH (δ)	9.4	39.5	48.8	55.8	67.4
H	9.2	25.6	44.2	55.8	46.5
WH	.8	25.6	34.9	30.2	44.2
SH	1.9	20.9	41.8	27.9	32.5
V	.7	11.6	25.6	23.2	32.5

*These percentages refer to the percentage of occurrence of the specific sound in 573 sounds in each reading selection. These 573 sounds include initial and medial consonants and initial vowels.

sounds listed is the same as that in Table I, based on the median percentages. Thus the percentage of occurrence of a sound and the percentage of stutterers having difficulty with a sound can be compared in the light of the suggested rank of order of consonants. The sounds which occurred most frequently in the reading material, i.e., TH (voiced), S, H, W, F, and P, were *not* sounds upon which stuttering occurred most often. The percentage of occurrence of a sound then has no appreciable effect on the ranking of difficulty of sounds.

Mention has been made above in the discussion of procedure of the fact that stuttering occurred on medial consonants in twenty-three words. In the majority of these words the consonants with which stuttering was associated began an accented syllable. Brown's study (2) on stuttering in regard to accent found that the greater percentage of stuttering occurred on accented first syllables, and that stutterers showed a greater tendency to stutter on accented second and following syllables than on unaccented syllables. This present study agrees with Brown's findings. In the forty-three cases considered, thirty stutterers experienced spasms on medial consonants. Of the thirteen who did not show such tendencies, ten stuttered only slightly and therefore do not influence the conclusion that many stutterers experience spasms on accented medial consonants. However, only 1.87 per cent of the total number of stuttering spasms of the forty-three stutterers occurred on the medial consonants. The other 98.13 per cent of the stuttering occurred on initial sounds. Brown (2) found that 92 per cent of the spasms of his group occurred in relation to initial sounds.

There were some atypical individual patterns in stuttering. For instance, Case 11 stuttered on the R in "dropped" in Situations 2 and 3; Case 17 stuttered on the unaccented M in "German" and the K in "market," and prolonged the usually unaccented second A in "valuable." Case 27 three times blocked on the P in "spoke," and Case 42, a pronounced stutterer, besides having difficulty on all consonants and on vowels, exhibited such patterns as "Chester, prisoner, difficulty, glittering, five, see, have, lonely, mother." (The sounds upon which stuttering occurred are in bold type.) Another case, 17, appeared at first consideration to be stuttering on final consonants. He prolonged the final consonant or vowel when it was a continuant or held the speech mechanism in position on a final plosive. Finally, he would produce a short "uh" sound and speak the next word explosively. For example, "Five minutes-s-s-s (ʌ) later, "A good (tongue remaining tensely against teeth ridge) (ʌ) show," "Lonely-e-e-e (ʌ) prisoner." Upon being questioned, he admitted that he prolonged the final sound to break the block in the initial sound of the next word.

Of the 124 initial vowel sounds occurring in each of the four reading selections, or a total of 496 vowels, the forty-three stutterers totaled as a group had spasms on 610 vowels. Thus it was suggested that stuttering occurred on 2.9 per cent of the vowels in the four reading selections. However, these figures are not conclusive, as it must be recalled that differences in situations affected the test results. Johnson and Brown's study (4) had also established that stuttering occurred predominantly on consonants.

One stutterer, Case 31, displayed a most peculiar stuttering pattern. His overt stuttering seemed to occur on short words: articles, pronouns, and prepositions. Thus a great deal of stuttering occurred on the article "a," on the conjunction "and," and on pronouns and prepositions starting with vowels. He also had trouble on longer "vowel-words." Ignoring the variation in number of spasms associated with changes in situation and summing up his responses to the four situations, it was found that 40.5 per cent of his stuttering occurred on vowels, or that he stuttered on 28.1 per cent of the vowels in the text.

In spite of the fact that a ranking of difficulty of sounds related to stuttering occurrence is offered, the records of the individual cases showed that the stutterers differed to a marked degree both on the sounds upon which stuttering occurred, and in the amount of difficulty with specific sounds. The Johnson-Brown study (4) found that individual rankings of difficulty for sounds may differ greatly from the group ranking, and that the individual factor was far stronger than the general factor in influencing rankings of difficulty for sounds.

In analyzing individual cases it was found that out of the forty-three stutterers, the data on seventeen indicated significant difficulty with specific sounds. Twenty-two did not stutter sufficiently to show that one sound appeared more difficult than another for them. Four pronounced stutterers distributed their stuttering with a certain amount of equality over all sounds. The difficulty of the seventeen was analyzed on the basis of the percentage of the occurrence of each sound in the reading selections. If a case stuttered on 25 per cent or more of the number of times a sound occurred, and if the data on that case showed that these sounds predominated others in stuttering, the data was said to suggest actual phonetic difficulty. Those cases which gave evidence of such difficulty are listed:

Case 1: P, B, T, D, K, G, CH, J
Case 2: P, B, T, D, G
Case 3: D, G, L, CH, J
Case 4: G, N, L, R
Case 8: D, CH
Case 9: G, N
Case 12: W, S
Case 13: K, T, R, J
Case 14: R
Case 20: M, N, R, L, W, CH, J, TH (unvoiced)
Case 22: P, D, G, N, M, R, L, W, TH (unvoiced)
Case 23: P, D, G, N, R, L, CH, J
Case 25: P, F, S, SH, TH (unvoiced)
Case 26: D, M, N, G, R, L, CH
Case 27: B, D
Case 31: (Vowel stuttering), TH (voiced), H
Case 34: G, R

Case 15 stated to the tester that he had always had great difficulty with the initial R in stuttering. Yet his tests display no such difficulty. Case 25 is exceptional in that he had the greatest difficulty with the voiceless sounds. All but two of these seventeen subjects showing difficulty on certain sounds were pronounced stutterers. Johnson and Brown (4) found that the more severe the stuttering the more likely was one to be consistent in stuttering on certain sounds. The analysis showed that although a ranking of sounds associated with stuttering could be suggested, the individual variations were far more pronounced than the group tendency toward formulation of such a ranking.

Summary

Of the fifty-two adult stutterers who were tested in this investigation, forty-three completed the four varied reading situations. The source of data for this space of the study is the same as that reported in a previous article by the author (3). These forty-three stutterers each read 550-word reading selections in four socially varied situations. A concealed observer marked the occurrence of each spasm in the reading material, thus reporting for each case the sounds associated with stuttering. The 550-word reading selections were analyzed phonetically to establish the number of times each consonant occurred initially. Since it was found that twenty-three medial consonants were associated with stuttering, these were added to the list of consonants. The number of initial vowels was also ascertained.

On the basis of this study the following conclusions on the relationship between the phonetic factor and stuttering occurrence are offered:

1. It is possible to arrange the sounds in a ranking of difficulty according to median and mean per cent of stuttering experienced in relation to each sound. The five sounds associated with the greatest amount of stuttering are: G, D, TH (unvoiced), L, and CH.

2. This ranking of difficulty can be compared with that of Johnson and Brown (4) with fair correlation. The G, D, L, TH (unvoiced), CH, and M in the larger percentages, and the F, S, SH, WH, TH (voiced), W, and H in the smaller percentages are relative in position in the respective tables.

3. Although a general ranking of difficulty of sounds can be set up for the group, individual stutterers vary widely on sounds associated with stuttering and in the amount of stuttering on specific sounds. In this study seventeen of the forty-three stutterers tested showed predominance of certain sounds over others in their stuttering.

4. The ranking of difficulty of sounds can not be said to show the influence of physical factors in sound formation. Voiced and voiceless, plosive and continuant classifications, or the location, direction, and duration of movements in sound formations, seem to have little bearing on the formulation of the general ranking of difficulty of sounds in stuttering. The difficulties of individuals also do not fall into such classifications of sounds.

5. Stuttering occurs more predominantly in consonants than on vowels.

6. The preponderance of stuttering occurs on initial sounds. The majority of medial consonants associated with stuttering are at the beginning of the accented syllable wherein the stuttering occurs.

Since the phonetic factor in the stuttering of the group is not a strong influence, it is suggested that the physical element in the production of sounds has little relation to stuttering occurrence. Other factors must be operating, the most likely of which is the psychological. The data in this particular study do not objectify psychological factors. In continuing the study of the data furnished by the tests of these stutterers, the grammatical factor and its relationship to stuttering occurrence merits attention.

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THE FREQUENCY OF STUTTERING IN RELATION TO WORD LENGTH DURING ORAL READING *

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Introduction

In 1935 Johnson and Brown demonstrated that there is a factor of phonetic difficulty which influences the loci of stuttering spasms in the oral reading situation (1). Later, Brown found that there exists a grammatical factor of difficulty, since certain parts of speech are more likely to be stuttered (2), and that syllable accent and word position are also important in determining the loci of stuttering (3).

Among other factors which could conceivably influence the location of spasms is that of word length. The present study was undertaken in an attempt to determine whether word length as such is one of the factors of importance in locating spasms at various points in the speech sequence.

The only previous investigation which relates directly to the present problem is an unpublished study by Milisen, who found the frequency of stuttering to vary directly with word length. However, Milisen used single words which were shown by means of an exposure device. The subject was permitted to see the word for a moment, and was then to indicate whether he expected to stutter when he said the word. At a signal from the experimenter, the stutterer attempted the word, and the experimenter recorded whether or not stuttering occurred. The situation was thus a highly specialized laboratory situation, with responses made in single words rather than in connected, meaningful speech. Milisen also had fewer subjects than the number used in the present study.

Procedure

"Thirty-two stutterers served as subjects in the collection of the data. Each of these read approximately 10,000 words of material to the experimenter. The material was composed of five 1,000-word reading selections, each of which was read twice by each case. The material was simple in construction and thought, and was entirely factual in nature. The experimenter recorded on a mimeographed copy of the selection being read all stuttering spasms, reading errors, mispronunciation, and variant pronunciations. In a few cases the amount and severity of stuttering were so great that the subject was unable to complete the reading. Such instances were few, however, and their occurrence did not prevent the collection of adequate data for any subject.

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*This study is a part of the program of research of the University of Minnesota Speech Clinic, Professor Bryng Bryngelson, Director. The complete original data are on file at the Clinic.

"The reliability of the experimenter[1] was determined by correlating his marking of spasms from a phonograph record of stuttering speech with the count known to be correct, and was found to be .99." (4)

Since one of the studies previously referred to had demonstrated the existence of a grammatical factor of difficulty, this factor was held constant by limiting the study to adjectives, which were the most difficult part of speech, and prepositions, which were next to the easiest.

Two measures of word length were used, the number of syllables and the number of letters, and analyses were made in terms of both of these. For every case the percentage of stuttering on each letter-syllable group of adjectives and of prepositions was determined. The mean of these percents of stuttering was then found for the entire group of thirty-two cases for each letter-syllable category.

The phonetic difficulty of all words in each group was determined from data given in the Johnson-Brown study (1), and the average phonetic difficulty for each letter-syllable group was then found. These measures of phonetic difficulty for each of the groups were correlated with the mean percents of stuttering on each letter-syllable group by the method of rank-differences.

Results

The average number of syllables per word for all words in the five reading selections is 1.46. The average number of syllables for adjectives is 1.65, and for prepositions is 1.13. The average number of letters per word shows the same trend, with 4.51 being the average for all words, 5.31 for adjectives, and 2.75 for prepositions.

For adjectives, there is a general trend for the mean percent of stuttering to vary directly with the word length, but the variation shown in Table I is irregular. If the one-syllable adjectives which are least difficult are compared with the two-syllable adjectives which were stuttered least, the two-syllable group shows a higher percent of stuttering. The comparison between the lowest means within the two- and the three-syllable groups show similar results. If the highest means within each syllable category are compared, the two-syllable adjectives are more difficult than the one-syllable group, and the three-syllable are stuttered oftener than the two-syllable adjectives. It should also be pointed out, however, that within any syllable group there is not a regular increase in the percent of stuttering with an increase in the number of letters in the word.

The longest adjectives in both the one- and the two-syllable groups shows a higher percent of stuttering than the shortest words in each group, but the nine-letter three-syllable adjectives are easier than the seven-letter three-syllable adjectives.

In every case, the standard deviation of the distribution is almost as large as, or larger than, the mean percent of stuttering on the corresponding word length group, indicating a great deal of variation from one individual to another. None of the differences between any two

[1]A fuller discussion of this method of determination of reliability is found in Johnson and Brown, *loc. cit.*

TABLE I

STATISTICAL MEASURES OF STUTTERING ON SYLLABLE-LETTER GROUPS OF ADJECTIVES AND PREPOSITIONS FOR 32 CASES

Syllable-Letter Group	Phonetic Difficulty*	Mean % of Stuttering	S. D.	Median % of Stuttering	Range
Adjectives:					
1 Syll.—2 L	12.0	11.78	15.53	7.74	0-62.50
3 L	7.2	7.37	7.48	5.26	0-28.35
4 L	12.5	12.55	13.79	4.50	0-53.87
5 L	12.6	12.43	13.63	6.37	0-48.97
2 Syll.—4 L	5.6	8.09	7.53	5.88	0-27.94
5 L	7.0	9.67	8.29	8.19	0-27.02
6 L	9.7	16.51	14.65	11.11	0-42.22
7 L	11.1	15.06	14.28	9.41	0-43.58
8 L	12.9	15.00	14.69	10.18	0-46.29
3 Syll.—7 L	8.9	17.99	19.29	6.25	0-55.00
8 L	13.2	22.53	21.72	13.02	0-61.76
9 L	10.1	15.92	16.77	9.00	0-53.33
Prepositions:					
1 Syll.—2 L	3.6	2.36	3.28	.91	0-13.36
3 L	10.2	4.44	7.13	1.56	0-35.86
4 L	10.5	6.33	7.87	3.38	0-36.17
2 Syll.—4 L	3.9	3.88	6.62	0.00	0-28.57
5 L	1.3	5.83	7.01	1.25	0-25.00
6 L	11.0	11.91	12.95	6.94	0-40.62

successive syllable-letter groups, or between two groups of the same number of letters but a different number of syllables, was such as to meet even the ten percent level of significance. Hence no table of these results is included.

Table I also contains data for prepositions similar to that for adjectives, with somewhat comparable results being found. Again a general rise in mean percent of stuttering accompanies increase in syllable length. Within each syllable group the percent of stuttering varies systematically with the number of letters in the word.

The standard deviations are large for every syllable-letter group. Some of the differences between means were such that the application of Fisher's t-test[2] showed them to meet the five or one percent levels of significance. The t-value of the difference between mean percents of stuttering on five- and six-letter two-syllable prepositions satisfies the five percent level of significance, and the differences between two-letter one-syllable and four-letter one-syllable, and between four- and six-letter two-syllable prepositions are significant at the one percent level.

The data were regrouped in Table II to show the difficulty for all

*This refers to the average difficulty for these same 32 cases of the initial sounds of the words in each syllable-letter group. The values for phonetic difficulty were computed from data given in the Johnson and Brown study (1, p. 491).

[2]The data were analyzed statistically according to Fisher's formulae as found in Lindquist, E. F., *Statistical Analysis in Educational Research*, Houghton Mifflin, New York, 1940.

words of each syllable and each letter length. Thus the one-syllable adjective group contains all one-syllable adjectives regardless of number of letters. Three-letter or four-letter groups include all adjectives of this letter length, irrespective of the number of syllables per word. The same is true for the prepositions. When the data are grouped in this manner, a steady increase in the mean percent of stuttering is found to accompany an increase in syllable length. A similar but irregular increase is noticed in letter length categories.

Comparisons between the difficulty found for each syllable or letter group were made with all other syllable or letter classifications, but only those which satisfy the ten percent or higher level of significance are included in the table.

Although there is a steady increase in the mean percent of stuttering with adjectives of more syllables, the application of the t-test to the differences between the one- and two-syllable adjectives, and between three- and four-syllable adjectives yielded values which did not satisfy the ten percent level of significance. The t-values of the differences between three-letter adjectives and each of the six- through ten-letter

TABLE II

MEAN PERCENTAGES OF STUTTERING FOR SYLLABLE AND LETTER CLASSIFICATIONS OF ADJECTIVES AND PREPOSITIONS WITH MEASURES OF THE SIGNIFICANCE OF DIFFERENCES

	Mean % of Stuttering	Comparison between average %'s of Stuttering for Groups	t-values	Percent Level of Significance
Adjectives:				
No. Syllables				
1	11.41	1 and 3	1.87	10
2	12.61	1 and 4	1.90	10
3	18.86	2 and 3	1.59	10
4	19.41	2 and 4	1.65	10
No. Letters				
3	6.43	3 and 4	2.05	5
4	11.60	3 and 5	2.14	5
5	11.54	3 and 6	3.42	1
6	16.65	3 and 7	2.84	1
7	14.85	3 and 8	3.43	1
8	18.17	3 and 9	2.97	1
9	16.09	3 and 10	3.12	1
10	17.86	4 and 8	1.72	10
		5 and 8	1.77	10
Prepositions:				
No. Syllables				
1	3.19	1 and 2	2.46	1
2	6.98			
No. Letters				
2	2.36	2 and 4	2.47	1
3	4.44	2 and 5+	3.53	1
4	5.57	3 and 5+	1.98	5
5+	8.58			

groups meet the one percent level of significance. The comparisons between four- through ten-letter adjectives with those of all other lengths, however, yielded t-values so small that even the ten percent level was not reached. The only exceptions are those differences between four- and five- as compared with eight-letter adjectives.

When one- and two-syllable prepositions are compared, a t-value is obtained which satisfies the one percent level of significance. Two-letter preposition, when compared with four- and five-and-more-letter groups, show equally significant differences. The difference between three-letter prepositions and five-and-more-letter prepositions is significant at the five percent level.

The rank-order correlations between the mean percent of stuttering on syllable-letter groups and the mean phonetic difficulty for the corresponding groups are as follows: for adjectives the correlation is .48, with twelve pairs of variables; for prepositions it is .68, with six pairs of variables.

Discussion and Summary

The differences noted when comparisons are made between the amounts of stuttering on words of various lengths clearly indicate that there is a tendency for stuttering to vary directly with word length. Although there are several instances in which the variations might be considered to show the opposite trend, many of those differences can be explained on the basis of the average phonetic difficulty for the words in those categories (see Table I). For example, four-letter two-syllable prepositions were stuttered 3.88 percent of the time, whereas four-letter one-syllable prepositions show a mean percent of stuttering of 6.33, and three-letter one-syllable prepositions a mean of 4.44. When the average phonetic difficulties of these groups are compared, three- and four-letter one-syllable prepositions were found more difficult, with 10.2 and 10.5 being the average phonetic difficulties. The phonetic difficulty for four-letter two-syllable prepositions was only 3.9. Some of the other irregularities in the variation of stuttering with respect to word length are also to be explained on the basis of relative phonetic difficulty.

The correlations of .48 and .68 between the amounts of stuttering on adjectives and prepositions of different word lengths with the average phonetic difficulty of the corresponding groups indicate that to a certain extent the variations in stuttering on syllable-letter groups may be accounted for on the basis of differences in phonetic difficulty. However, the lack of a high relationship even when measured by the method of rank-differences indicates that the tendency for longer words to be stuttered more is not due solely to phonetic difficulty. Since grammatical function was held constant in these comparisons, and since syllable accent would not seem to be a factor to consider here, it seems justifiable to infer that word length is an important factor in the differences found.

It is impossible to determine whether the number of letters or the number of syllables in a word is the better measure of the relative difficulty it is likely to present for stutterers. Moreover, even if the

present data could yield such information, there would be a relatively small difference in the importance of one of these measures as opposed to the other, inasmuch as there is a very strong tendency for them to vary together. There is a more regular rise in the percent of stuttering from one syllable group to another than from one letter group to another, but more variation would be expected in letter comparisons because of the greater number of categories. There is a regular rise in mean percent of stuttering with an increase in the number of letters within each syllable group of prepositions. This can not be explained on the basis of phonetic difficulty. In the case of adjectives the same phenomenon, although irregular, is nevertheless striking and again can not be explained solely on the basis of phonetic difficulty. From these facts, word length as such would seem to be a factor in determining the loci of stuttering spasms.

Since adjectives have been shown to be the most difficult part of speech, and prepositions next to the easiest part of speech (2), it seems reasonable to assume that the results of the comparison made would also hold true for other parts of speech.

If spasms were distributed on an absolutely random basis throughout the speech sequence, longer words would probably be stuttered more frequently than shorter words because of their longer duration in time, thereby presenting more opportunity for stuttering. However, it has been demonstrated that factors other than chance operate in determining the loci of stuttering spasms (1, 2, 3, 4). Hence it becomes necessary to account for the greater percentage of stuttering on longer words in terms of other factors.

Longer words, regardless of their grammatical function, are more prominent in the speech sequence because of their greater duration in time, and it is logical to assume that for this reason stutterers would place a greater premium on free speech in relation to them. It has been shown by Steer and Johnson that the amount of stuttering varies directly with the stutterer's desire for free speech (5). This factor is probably the most important one in causing longer words to present more difficulty for stutterers than do shorter words.

A number of other psychological factors may operate to make longer words more difficult for stutterers than shorter words, but the one just mentioned is the most obvious, and likely more important than all the rest.

Summary and Conclusions:

A statistical study was made of the relative frequency of stuttering in relation to adjectives and prepositions of various lengths. Word length was measured both in terms of letters and of syllables. Thirty-two stutterers served as subjects and read passages to an experimenter.

The following conclusions may be drawn from the data:

1. Longer adjectives and prepositions are more difficult for stutterers than are shorter adjectives and prepositions, whether syllable-letter, syllable, or letter categories are used as a basis for measuring word length.

2. The factor of phonetic difficulty explains some of the above relationships, but not all of the variation can be explained in this way.
3. From these data, it seems reasonable to conclude that, other factors being equal, the amount of stuttering tends to vary directly with word length.
4. A possible explanation for this is found in terms of the greater prominence of longer words and the stutterer's consequent greater desire to avoid stuttering at those points.

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REPORT OF A SPEECH SURVEY, HOLYOKE, MASSACHUSETTS

ALICE W. MILLS AND HELEN STREIT

Introductory note. The survey here reported was undertaken at the request of the Superintendent of Schools for the purpose of securing data which might be a determining factor on the question (which had been under consideration for some months) as to the advisability of appointing a speech correctionist to the city school system. The resultant facts and figures served to convince the school board of the need in this direction, and within two months after our report was turned in a full-time position in speech corrective work was created. Miss Marion Kennedy, of Holyoke, who had assisted in the survey, was given the appointment. We hope that when the investigation is completed and the full significance of the speech situation in the Holyoke schools is made clear, a further appointment will be made and that the teachers will be required to take some basic training in speech behavior in order that they may cooperate more efficiently with the speech correctionist.

We offer our findings to date for publication in the belief that our figures may have some value in relation to similar surveys, and also because the continuation of the investigation is being delayed owing to the preoccupation this year of all the examiners in national defense projects. It seems to us, in view of the uncertainty of completion of this survey within the near future, unwise to leave our findings unpublished.

During the school year 1940-1941, a study was made of the speech of forty-six hundred and eighty-five (4685) children in the schools of Holyoke, Massachusetts. This project grew out of the speech corrective work in the Holyoke Child Guidance Clinic—work which was started over twelve years ago by Dr. Sara Stinchfield and carried on for the six years just preceding the survey by Professor Alice W. Mills of Mount Holyoke College. During these years the cases of speech defective children referred to the clinic from the schools increased to such an extent that it became impossible to take care of them in a clinic that had not been instituted primarily for this purpose. The teachers became interested in the possibilities for meeting speech problems in the schools and the superintendent, having for some time wished for data on the subject, was ready to initiate the plans and cooperate in a survey.

Objectives

The primary objectives of the investigation were first, to discover the number of children with speech handicaps of a nature serious enough to call for treatment by a speech pathologist, and second, the number with less serious defects who might be taken care of by classroom teachers having basic knowledge of speech functioning and speech problems. Obviously some preliminary consideration of etiological factors was necessary in order to make possible this classification, and in following this procedure it was found advisable to give some speech training during the building of case histories, as, with this approach, it was easier to get at diagnoses. Thus the original objectives widened in scope and significance.

Procedure

There were ten examiners in all; they were not equally well prepared for the work, but all of them had had at least one year of training under recognized experts in the field of speech correction and six of them had studied under three to four authorities in speech pathology. During the period of the survey the examiners met frequently to discuss individual cases, methods of investigation, standards of judgment, terminology and unforeseen problems.

A close cooperative relationship established at the start with principals, teachers, and the school physician proved of great value. The director of the survey enlisted the cooperation of all the school principals in private conference, and explained the whole project in detail to all the teachers, meeting them in small groups in order to more surely stir them to active response. The results of these conferences were evidenced in many ways. Teachers showed themselves not only willing and even anxious to assist us in any possible way, but developed some ability in recognizing speech defects. We were enabled to consult the school physician at any time, to consult medical and family histories, audiometer tests and school reports, to arrange for intelligence tests, to observe children in the classroom and on the playground, to call a child from the classroom for individual testing or training at any time of the day, and to arrange (through the principal) for conference with parents. Without this whole-hearted cooperation and the deep interest and support of Supt. Wm. Pack, it would not have been possible to accomplish the results.

We tested first the cases that had been recognized and referred by the teachers, and next all the children in the first three grades. By the time we had progressed this far other cases had been discovered by the teachers and these were examined. Following this procedure, children in some classes of the upper grades were checked in classroom recita-

tion; however, time did not permit all upper grades to be visited nor all children in upper grades to be given individual tests, and therefore we can give statistics for only the first, second and third grades, the cases referred by teachers, and the cases discovered by the examiners in visiting classes—since only these children (twelve hundred and seventy in all) were given individual testing. It is to be noted, however, that our figures represent the total school enrollment, viz. forty-six hundred and eighty-five (4685) children, since the referred cases and the cases discovered in class visiting came from all school grades.

Each child from the three groups (viz. the referred cases, the "discovered" cases and the first three grades) was tested in individual conference, and by at least two examiners. Occasionally a third examiner was called in on a case. These conferences were held in the school buildings during regular school hours in order to keep the situation as familiar for the child as possible. All children old enough to read were tested in both reading and spontaneous speech. Pictures, charts and games were used to elicit speech patterns representative of all speech sounds and of difficult sound combinations. When a defect was immediately observable, the examiner followed this lead to a completed check; in other cases—where the defect was not prominent or instantly evident—the examiners followed a pre-determined order of procedure. General body coordination was observed, speech structures examined when occasion demanded, reference made to data in school files, (I.Q., audiometer, medical, etc.), occasional visits made to the home (an invitation to drive the child home after school being given as the excuse), and rechecks made on every child who had seemed during the test frightened, nervous or ill. Forms for the recording of preliminary data on all children and for full case histories on serious cases were generously furnished by the school system.

Results

The following figures represent our findings. Since *every* child was tested in only the *first three grades,* we give separate figures on these results. If we had forseen the impossibility of completing all grades in individual testing during the year, we should not have started with the first lower grades. Obviously a large percentage of the articulatory defects in the younger children will disappear with the maturation process. However, we offer these figures as further data on the phases of the general problem to which they relate.

TOTAL NUMBER OF SPEECH DEFECTIVE CASES AMONG 4685 CHILDREN

Type of defect	Grades: First three (1196 children)		Above third (3489 children)		Total (4685 children)	
	no.	percent	no.	percent	no.	percent
Dyslalia	296	24.7	30	.9	326	7.0
Dysphonia	67	5.6	5	.1	72	1.5
Dysphemia	32	2.7	37	1.1	69	1.5
Cleft Palate	2	.2	2	.05	4	.09
Slobbering	1	.1	—	—	1	.02
Mutism (Hysterical)	1	.1	—	—	1	.02
Total	399	33.4	74	2.1	473	10.1

NUMBER AND PERCENTAGE OF 473 SPEECH DEFECTIVE CASES CLASSIFIED AS SERIOUS

Type of defect	Grades: First three (399 cases)		Above third (74 cases)		Total (473 cases)	
	no.	percent	no.	percent	no.	percent
Dyslalia	89	22.3	23	31.1	112	23.7
Dysphonia	32	8.0	5	6.8	37	7.8
Dysphemia	26	6.5	31	41.9	57	12.1
Cleft Palate	2	.5	2	2.7	4	.8
Slobbering	1	.3	—	—	1	.2
Mutism	1	.1	—	—	1	.02
Total	151	37.8	61	82.4	212	44.8

151 is 12.6% of enrollment of first 3 grades (1196).
61 is 1.7% of enrollment of grades above 3rd (3489).
212 is 4.5% of total school enrollment (4685).

NUMBER AND PERCENTAGE OF 473 SPEECH DEFECTIVE CASES LISTED AS SERIOUS DYSLALIA CASES, CLASSIFIED ACCORDING TO TYPE OF ARTICULATORY DIFFICULTY

Type of articulatory defect	Grades: First three (399 cases)		Above third (74 cases)		Total (473 cases)	
	no.	percent	no.	percent	no.	percent
Sigmatism	56	14.0	10	13.5	66	13.9
r	1	.3	1	1.4	2	.4
l	3	.8	—	—	3	.6
th	6	1.5	1	1.4	7	1.5
Other consonants	1	.2	1	1.4	2	.4
Mult. art.	22	5.5	10	13.5	32	6.8
Total	89	22.3	23	31.1	112	23.7

NUMBER AND PERCENT OF SPEECH DEFECTIVE CASES IN GRADES 1, 2, AND 3

Type of defect	Grades							
	1 (456 enr.)		2 (347 enr.)		3 (393 enr.)		Total (1196 enr.)	
	no.	percent	no.	percent	no.	percent	no.	percent
Dyslalia	132	28.9	77	22.2	87	22.1	296	24.7
Dysphonia	20	4.4	24	6.9	23	5.9	67	5.6
Dysphemia	7	1.5	15	4.3	10	2.5	32	2.7
Cleft Palate	—	—	1	.3	1	.3	2	.2
Slobbering	1	.2	—	—	—	—	1	.1
Mutism	1	.2	—	—	—	—	1	.1
Total	161	35.3	117	33.7	121	30.8	399	33.4

NUMBER AND PERCENT OF CASES LISTED UNDER DYSLALIA IN GRADES 1, 2, AND 3

Type of articulatory defect	Grades							
	1 (456 enr.)		2 (347 enr.)		3 (393 enr.)		Total (1196 enr.)	
	no.	percent	no.	percent	no.	percent	no.	percent
Sigmatism	64	14.0	36	10.4	49	12.5	149	12.5
r	4	.9	5	1.4	5	1.3	14	1.2
l	7	1.5	6	1.7	3	.8	16	1.3
th	35	7.7	19	5.5	20	5.1	74	6.2
Other consonants	3	.7	2	.6	2	.5	7	.6
Mult. artic.	19	4.2	9	2.6	8	2.0	36	3.0
Total	132	28.9	77	22.2	87	22.1	296	24.7

SEX DIFFERENCES, FIRST THREE GRADES, 399 CASES

Type of defect	Sex				Total	
	Boys		Girls			
	no.	percent	no.	percent	no.	percent
Dyslalia	184	46.1	112	28.1	296	74.2
Dysphonia	39	9.8	28	7.0	67	16.8
Dysphemia	27	6.8	5	1.3	32	8.0
Cleft Palate	2	.5	—	—	2	.5
Mutism	1	.3	—	—	1	.3
Slobbering	1	.3	—	—	1	.3
Total	254	63.7	145	36.3	399	—

SEX DIFFERENCES IN ARTICULATORY DEFECTS AMONG 399 CHILDREN IN FIRST THREE GRADES

Type of articulatory defect	Sex				Total	
	Boys		Girls			
	no.	percent	no.	percent	no.	percent
Sigmatism	77	19.3	72	18.0	149	37.3
r	10	2.5	4	1.0	14	3.5
l	11	2.8	5	1.3	16	4.0
th	60	15.0	14	3.5	74	18.5
Other consonants	4	1.0	3	.8	7	1.8
Mult. Art.	22	5.5	14	3.5	36	9.0
Total	184	46.1	112	28.1	296	74.2

SEX DIFFERENCES ABOVE 3rd GRADE, 74 CASES

Type of defect	Sex				Total	
	Boys		Girls			
	no.	percent	no.	percent	no.	percent
Dyslalia	21	28.4	9	12.2	30	40.5
Dysphonia	4	5.4	1	1.4	5	6.8
Dysphemia	31	41.9	6	8.1	37	50.0
Cleft Palate	1	1.4	1	1.4	2	2.7
Total	57	77.0	17	23.0	74	—

SEX DIFFERENCES IN ARTICULATORY DEFECTS AMONG 74 CASES ABOVE 3rd GRADE

Type of articulatory defect	Sex				Total	
	Boys		Girls			
	no.	percent	no.	percent	no.	percent
Sigmatism	9	12.2	5	6.8	14	18.9
r	1	1.4	—	—	1	1.4
l	—	—	—	—	—	—
th	2	2.7	—	—	2	2.7
Other consonants	1	1.4	—	—	1	1.4
Mult. art.	8	10.8	4	5.4	12	16.2
Total	21	28.3	9	12.2	30	40.5

SEX DIFFERENCES

First three grades:

About ⅔rds of all cases are boys.

⅔rds of serious cases are boys.

Boys exceed girls in all classifications.

Greatest difference is stuttering. There are more than 5 times as many cases of boy stutterers as girls. (27 boys to 5 girls)

Least difference is voice. (39 boys to 28 girls)

In articulatory defects, boys exceed girls in all categories. Least difference is in sigmatism. (77 boys to 72 girls)

Above 3rd grade:

Over ¾ths of the referred cases are boys. (57 boys to 17 girls)

Over ¾ths of serious cases are boys.

Greatest difference is stuttering. There are slightly more than 5 times as many boys who stutter as girls. (31 boys to 6 girls)

Boys excell girls in all categories.

STUTTERING

69 cases out of total enrollment of 4685 or 1.5%.

In first 3 grades, 32 cases out of enrollment of 1196 children, or 2.7%.

Largest percentage was in second grade. 15 children or 4.3% of 347 children in second grade stuttered.

Smallest percentage was first grade. 7 children or 1.5% of 456 children enrolled in first grade were found to stutter.

In third grade 2.5% (10 out of 393) stuttered.

Above 3rd grade, 37 out of enrollment of 3489 were reported as stuttering, or 1.1%.

Sex differences. 5 times as many boys stutter as girls. (58 boys to 11 girls.) No difference shown above and below 3rd grade.

NOTES ON INTERESTING CASES

The case of hysterical mutism was a boy in the fifth grade who had a record of never having spoken in the classroom during his years at school. His fifth grade teacher was gaining his affection and confidence and expected to lead him to a change of attitude toward the school situation. The most obvious symptom of this case was his dislike (freely expressed to the teacher out of the classroom) of the other children. Another interesting articulatory case was of a lip-tie; the upper lip seemed to the examiners to be anchored to the upper gum at a point lower than the normal, or average.* One articulatory case, reported by the teacher as serious, proved to be a "gum-hider." According to his own statement, he kept a wad of gum sticking to the arch of his palate (which was high) at all times, day and night—even when eating. He spoke without parting his teeth. When giving his confidence he bargained, "I'll tell you what it is if you'll promise not to tell on me." A further instance of confidence in the examiner was of a boy (who had not been reported by his teacher) who asked to see the examiner during recess. He said: "Don't you tell! But I stutter. Both my father and my brother stutter, too. I can hide it at school; but I wish you'd tell me how to stop it, cause I'd like to talk more." But perhaps the most interesting case of all was of a ten-year-old boy, who had never been sent to school. It is a case of idio-glossia, reported by a teacher who is a friend of the family. These, and other cases, offer worthwhile opportunities for further study.

*The school physician, however, did not consider the condition pathological.

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Enid—Phillips University—Morris, J. A. *Norman*—University of Oklahoma—Ritzman, C. H. *Oklahoma City*—Kelley, K. B.; Kimmell, M. L. *Stillwater*—A. and M. College—Voelker, C. H. *Tulsa*—Waddle, E. L.

OREGON:
Corvallis—State College—Wells, E. W. *Eugene*—University of Oregon—Bangs, J. L. *Portland*—Gardner, W. H.

PENNSYLVANIA:
Beaver Falls—Geneva College—McCalmon, G. A. *California*—California State Teachers College—Morely, D. E.; Pennsylvania State College—McDonald, E. T. *E. Stroudsburg*—State Teachers College—Barnett, R. *Harrisburg*—Westlake, H. *Johnstown*—

(Continued to page 171)

DISEASES OF THE UPPER AIR PASSAGES AS CAUSES OF HEARING DEFICIENCIES

By Russel G. Means, M.D.
Columbus, Ohio

At the beginning of this paper I will mention some mechanical hazards that must be considered before discussing the inflammatory disease conditions both of which imperil the hearing mechanism and are steps toward progressive deafness.

Accidents such as fractures of the base of the skull, not uncommonly result in damage to the delicate end organ of hearing. Avoidable contingencies which should be warned against are a blow or slap and, now less commonly encountered, the explosion of firecrackers or a gun near the ear.

Most industrial hazards now are being recognized and eliminated.

Since man has taken to the air, commercial as well as war flying presents perils to the delicate auditory organs. Rapid changes of altitude entail either increased or decreased air pressure. These insults traumatize and may cause serious hemorrhages or scar tissue formations. Flying is particularly dangerous to those with eustachian tube abnormalities or individuals suffering from infections of the nose or throat. Such repeated affronts of an extreme nature, encountered by flyers, lead to irrepairable lesions in the labyrinth.

Another abuse overlooked today, is that of uncontrolled and improper swimming which is creating a definite menace not only to the ears and sinuses but to the general health of many children.

Diseases or infections involving the ears and directly affecting hearing are of two forms; as stated by many authorities, including Dr. Gordon Berry.

First, severe toxic diseases whose poisons may affect the labyrinthine end organ, the acoutic nerve or injure the hearing centers in the brain. The most common offenders are Scarlet Fever, Meningitis, Mumps and Syphilis; others occasionally acting in this manner are Influenza, Diphtheria, Encephalitis and Measles. Also in this classification must be considered general depleted states or systematic diseases that weaken the whole body, starve the tissues and predispose the sufferers to intercurrent secondary infections, which in susceptible individuals causes deterioration of hearing. Physical states, such as anemia, fatigue, exhaustion, extreme exposure, lack of proper food and vitamins fall in this group. We must recognize that the undernourished, which includes the undisciplined cranky or "faddy" eaters, both children and adults, become prey to acute upper respiratory infections as well as insidious tissue starvation. Our well being, or

state of health, does not maintain an even level and, therefore, in the lives of nearly everyone, at sometime, it is necessary to be extremely careful to protect our health.

One drug, quinine, is notoriously damaging to the hearing apparatus and should never be used except to treat Malaria.

The second group, namely infections of the nose, throat and ear, offer the largest field for both the lay workers and practitioners of scientific medicine—those who hope to diminish the vast number of impaired hearing cases.

The common "cold" causes more loss of school and working days than any other disease. It also plows up the ground, so to speak, for subsequent complicating infections. The acute contagious infections of childhood: namely, Measles, Chickenpox, Diphtheria and Scarlet Fever begin with "cold" symptoms. Also Influenza, Sinusitis, Bronchitis, Pneumonia, Otitis-media (abscessed ears) frequently manifest the same symptoms. I must pause here to warn that all children should have the preventive innoculations against Smallpox, Diphtheria, Tetamus, Typhoid and Scarlet Fever. If the irrefutable mass of data accumulated by every army in the world has proven that these innoculations are absolutely essential, then everyone should surely take advantage of their God given, life saving properties.

Hereditary deafness can only be eliminated by the humanitarian principles of common sense and eugenics. Anatomically the ear is a well protected organ. However, man's natural enemies bomb and blitzkrieg our bodies and sensory organs resulting in permanent defects. To protect such victims requires intelligent recognition of these insidious and subversive disease enemies. Protective immunization against certain diseases will further reduce the number of impaired hearing cases. Yet, however cautious, the human flesh is subject to acute infections that may damage the ears and impair the hearing. If everyone would treat influenza, "colds," tonsillitis, sinusitis, et cetera, intelligently, by going to bed and obtaining competent medical care, we could still further reduce the number of cases of abscessed ears, mastoiditis or chronic sclerosing middle ear affections which are directly damaging to the hearing.

Obstructing or chronically infected tonsils and adenoids must be treated surgically. Nasal conditions such as sinusitis, whether acute or chronic, must be actively treated in order to preserve good hearing. If the parents, teachers or victims themselves, do not realize the dangers of the pathological conditions of the nose and throat, the services of the physician, surgeon, and oto-rhinolaryngologist are of little help. Delay in seeking this remedial attention will result in such damage as will lead to permanent auditory disability.

A new era has dawned with the discovery of sulfanilimide and its more recent improved derivatives. The administration of these miraculous drugs has practically eliminated the necessity for operations for acute mastoiditis. This rapid restoration of the ears to normality will

greatly reduce the number of hearing losses. I must caution that the use of these drugs are not without danger and require the supervision of a physician; otherwise relapses as well as masked complications may result.

I wish again to emphasize the fact that repeated or chronic nasal, pharyngeal or throat infections are always potential dangers and steps toward deafness. Sometimes after the tonsils and adenoids have been removed a subsequent infection in the nose or sinuses, as well as some allergic inflammations, may lead to a secondary growth of adenoid tissue on the lateral walls of the upper pharynx which blocks the openings of the eustachian tubes. Astute ear, nose and throat specialists are essential in order to diagnose and remove this tissue which directly causes reduced hearing.

Let me repeat that in order to get results in cases of progressive hearing loss, those afflicted with acute and chronic infections in the area adjacent to the ears must be sought out early through school hearing surveys, through education of the parents and the public. Medical attention in the early stages is absolutely essential if normal hearing is to be restored.

To be hard of hearing is not only a depressing affliction, but also a distinct economic handicap. If we are ever going to reduce the appalling number of otherwise useful citizens, who are burdened by deficient hearing, we must constantly carry on educational programs such as those which are being conducted by the Leagues for the Hard of Hearing. The causes of deafness must be impressed on the public in order to protect the oncoming generations from these potential dangers. Ignorance and superstition must be expelled or otherwise many innocent persons may suffer needlessly from loss of hearing, as many of you here can verify. Business men realize that when advertising is stopped, interest in their product declines and sales drop off. Everyone of us can do a great service to humanity by being a crusader to enlighten our school children, their parents and the general public about the care of their ears in order to decrease the number of individuals who may become afflicted by a hearing loss. DEFEND HEARING must be our watchword.

Delivered June 22, 1942—Cincinnati, Ohio
Central Zone Conference
American Society for the Hard of Hearing

1942 GEOGRAPHICAL DIRECTORY

(Continued from page 168)

Ellenberger, M. V. *Lahaska*—McDowell, E. D. *Lancaster*—Diller, G.; State College—Calvert, W. *New Wilmington*—Westminster College—Taylor, G. J. *Philadelphia*—McCausland, M.; Masland, M. W.; Nathanson, Y.; Nemoy, E. M.; Temple University—Tuthill, C. E.; University of Pennsylvania—Goodfriend, D. J.; Wea-

1942 GEOGRAPHICAL DIRECTORY

ver, H. B. *Pittsburgh*—Dick, A.; Levy, R. C.; Weiner, R. N.; Duquesne University—Golden, A. L.; Smith, F. *Secana*—University of Pennsylvania—Twitmyer, E. B. *Sharon*—Schafitz, E. R. *Slippery Rock*—State Teachers College—Backus, O. L. *State College*—Pennsylvania State College—Cobentz, I.; Harkins, C. S.; Koepp-Baker, H. *York*—Bell, A. R.

RHODE ISLAND:

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SOUTH CAROLINA:

Cedar Springs—Utley, J. L.

SOUTH DAKOTA:

Sioux Falls—Bennie, J. A.; Rugen, B. R.

TEXAS:

Austin—Pittinger, K. L. *Houston*—Knight, B. B. *Randolph Field*—Obermann, C. E. *San Angelo*—Compton, M. E.

UTAH:

Ogden—Russell, G. O. *Provo*—Brigham Young University—Morely, A. J.; Pardoe, T. E. *Salt Lake City*—Clark, R. M.

VIRGINIA:

Charlotteville—University of Virginia—Ansberry, M. *Lyncburg*—Adams, M. H. *Richmond*—Stoddard, J. E.

WASHINGTON:

Bellingham—Western Washington College—Cotton, J. C. *Ellensberg*—Central Washington College—Buck, M. S. *Seattle*—Jones, M.; Miller, E.; University of Washington—Carrell, M. A. *Tacoma*—Russell, L. M.

WISCONSIN:

Greenbay—Green, E. M. *Janesville*—Fishel, M. V. *Kanosha*—House, E. S. *Madison*—Camp, P. B.; Powers, M. B.; Ward, L. A.; Anderson, J. O.; University of Wisconsin—West, R. W. *Milwaukee*—Becker, R.; Duffey, W. R.; Templin, M.; Zimmer, C. H.; State Teachers College—Carr, A. M. *Racine*—Whitten, I. E. *Superior*—Steele, M. N.; Young, A. J. *West Allis*—Rutherford, V.

CANADA:

Saskatchewan—University of Saskatchewan—McConecky, K. M. *Toronto*—Berwick, N. H.; Dennison, W. *Montreal*—Huber, M. W.

SYMPOSIUM ON UNIQUE CASES OF SPEECH DISORDERS; PRESENTATION OF A CASE

By Henry M. Moser, Ph.D.
University of Michigan

This is a case of a young man 19 years of age with a very striking infantile voice. I have selected this case for presentation for several reasons. In the first place, the presentation may be made with phonograph records; second, that it presents an interesting combination of several principles of speech pathology, and finally, that it demonstrates the effectiveness of a method of voice training that I do not believe is well known.

I would like to play three records: first, the voice of this young man at the time he was admitted to the Clinic; second, the normal voice produced about an hour later, at the close of this record he will speak in the former voice for contrast. As will be explained later, the new voice was such a contrast to the old that he was unable to put it to use for a time. The third record was made three weeks later after he had succeeded in making the new voice habitual.

This patient is a nice looking young man of the long, lean asthenic type. He is of average height, considerably underweight and has a number of effeminate mannerisms. He is a junior college student, having been awarded a scholarship as the result of high scholarship in high school. All members of the family and known relatives have normal voices. Patient made these remarks about his voice: "I have always been sensitive because my voice set me apart from others. My teachers and classmates have always been considerate although school has never helped my problem. Even a course in public speaking failed to help in any way. I would describe it as an unusually light feminine voice of limited range, of little color, uninteresting, and of no ability to sing."

The mother did not believe the second record was her son until he demonstrated. After listening for a few minutes, she remarked, "Maybe it is too deep and big for such a skinny fellow."

Patient was informed of effeminate mannerisms and of possible difficulty of using this new voice in his environment. He was instructed to return to the Clinic at his earliest convenience for training. Patient returned home and used old voice for a week except when he was alone in his car. At the end of that time, he decided he 'had to make the leap' so he spent ten days with his grandmother in Canada during which time he used the new voice continually without a single failure. He returned to his home, used the voice for a week and then returned to the Clinic when this last record was made.

This is a case of a boy who, very probably sensitive to voice changes at puberty, made adjustments to keep his voice from breaking. These adjustments became habitual and resulted in a continued juvenile voice; that is, entirely inappropriate for his age and sex. Com-

bined with this, his extreme underweight, poor muscular development but superior intelligence have served to emphasize the juvenile voice and effeminate mannerisms.

The particular technique used to establish normal voice is the 'glottal fry' attack. It is a method I have used with considerable success and one which I do not believe is well known. It is very easy to demonstrate but extremely difficult to describe. You may recognize it as the sound produced by many youngsters in imitating a motor boat but to me it more nearly resembles the sound of vigorously popping corn. The patient produces this slow relaxed sound and by increasing the pressure of the air stream uses it as an attack to produce a low, full, normal tone.

CONSERVATION OF HUMAN RESOURCES IN WARTIME

Under the Auspices of

THE UNITED STATES EMPLOYMENT SERVICE
THE CALIFORNIA STATE BUREAU OF VOCATIONAL REHABILITATION
THE SOUTHERN CALIFORNIA SOCIAL SERVICE COUNCIL FOR THE HARD-OF-HEARING

Injuries sustained on the military and domestic fronts of the war are sure to undermine, to some extent, the nation's strength and morale. All public agencies concerned must take energetic steps to safeguard health and to procure prompt physical, emotional and vocational rehabilitation for the victims of modern warfare.

Hearing, so essential in human contacts, understanding and co-operation, is especially vulnerable in such times. Mechanized warfare and work in war industries, rampant disease and infection—measles, mumps, influenza, whooping-cough, common colds, infantile paralysis, spinal meningitis, diphtheria,—frequently result in impaired hearing. Therefore, we—doctors, nurses, parents, teachers, social workers, officers of school, health, rehabilitation and employment services, Red Cross, employers and workers—must mobilize our knowledge, our experience, effort and vigilence. We must safeguard the hearing and the health of our children, our soldiers, our workers and ourselves.

PROGRAM OF THE WORKSHOP
THURSDAY, JULY 2

8-9 —Registration at Harris Hall

9-12—Morning panels

GENERAL CHAIRMAN, Harry D. Hicker, *Chief California Bureau of Vocational Rehabilitation*

(Continued to page 178)

LARYNGEAL INFLAMMATION IN A CASE OF FALSETTO

By Barbara Allen and Gordon E. Peterson
Ball State Teachers College

This case entered the Speech and Hearing Clinic in April, 1942. The routine diagnostic examinations were begun on that date and were completed one week later. The following examinations were made:

A. Symptoms analysis:
   1. Nasality.
   2. Pitch.
   3. Quality.
   4. Flexibility.

B. Diagnostic analysis:
   1. Hearing.
   2. Nose.
   3. Throat.
   4. Motor abilities.
   5. A case history was taken, and a history of birth and early development was obtained from the parents.

The symptoms analysis showed the speech abnormalities listed below. All other speech skills appeared normal.

1. Falsetto voice with pitch at F below middle C.
2. Severe rhinolalia aperta on fricatives, semi-vowels, vowels, and diphthongs.

The diagnostic analysis showed the following possible etiological factors.

1. The lower right turbinate was considerably enlarged.
2. The hard palate was very high and the nasal septum was well deviated to the right.
3. The mucous membranes in both right and left nasal fossa were very inflamed.
4. A history of slight hay fever attacks was reported, but there had been no recent involvements.
5. The tonsils showed mild signs of pathology.
6. The vocal cords were considerably inflamed. The laryngeal examination, which was made at the same time that the nose was examined, is indicated below.

RECORD OF LARYNGEAL EXAMINATION PRECEDING SPEECH THERAPY

1 2 3 4 5 Defective Normal	Right		Left	
	Anterior	Posterior	Anterior	Posterior
.... Chorda vocalis				
Irregular	....	....	....	....
Inflammation	3	3	3	3
Growth	....	....	....	....
.... Thyro-arytenoideus				
Inflammation	3	3	3	3
Growth	....	....	....	....
.... Glottis				
Asymmetrical	....	....	....	....
Approximation				
Inadequtae	4	2	4	2
Over	....	....	....	....
.... Vocal process				
Inflammation		2		2
.... Arytenoids				
Inflammation		2		2
Enlarged		....		....

At the time of this examination the case reported that he had occasional colds, but that he had not suffered from one for several weeks. Beyond nose and laryngeal pathologies no other significant etiological factors were found, either from our examinations or from the case histories.

The recommendations made by the staff for the treatment of this case were as follows.

1. Medical referral for nose, throat, tonsils, and for a study of glandular development.
2. Begin pitch retraining immediately, with consistent observation of the effect of retraining on voice quality and on the condition of the larynx.
3. Treat for any remaining dysphonia and rhinolalia aperta after satisfactory pitch is established.

In the hope that lowered pitch would relieve strain and would thus facilitate improvement of the condition of the vocal cords during medical treatment, speech therapy was begun promptly. At the first meeting the procedure listed below was followed.

1. The nature of the problem and the possible associated causes were explained.

2. Medical examination and treatment were discussed.
3. The psychological problems involved in lowering pitch were explained.
4. Reading was practiced at falsetto and normal pitches.

The case returned for the second speech treatment two days later. He reported that following the first lesson he had found it so difficult to speak in falsetto that he had been using normal voice ever since. He was met five days later for a check and at that time a nasal examination showed no change in the condition of the nose. Though he reported that he was taking a cold, this could have merely been an impression resulting from his lowered voice. The accompanying chart is a record of the laryngeal examination made at this meeting.

RECORD OF LARYNGEAL EXAMINATION ONE WEEK FOLLOWING VOICE CHANGE

1 2 3 4 5 Defective Normal		Right		Left	
		Anterior	Posterior	Anterior	Posterior
....	Chorda vocalis				
	Irregular	....	....	....	....
	Inflammation	4	4	4	4
	Growth	....	....	....	....
....	Thyro-arytenoideus				
	Inflammation	4	4	4	4
	Growth	....	....	....	....
5	Glottis				
	Asymmetrical	....	....	....	....
	Approximation				
	Inadequate	....	....	....	....
	Over	....	....	....	....
....	Vocal process				
	Inflammation		4		4
5	Arytenoids				
	Inflammation		....		....
	Enlarged		....		....

Three days later a final examination was made. The voice quality was definitely pleasing, and the pitch, having been lowered from F below middle C to B below low C, appeared psychologically and physiologically optimum. The following chart is a record of the final laryngeal examination. Again there was no alteration in the condition of the nose.

RECORD OF LARYNGEAL EXAMINATION PRECEDING SPEECH THERAPY

1 2 3 4 5 Defective Normal	Right		Left	
	Anterior	Posterior	Anterior	Posterior
.... Chorda vocalis				
Irregular	....	....	....	....
Inflammation	3	3	3	3
Growth	....	....	....	....
.... Thyro-arytenoideus				
Inflammation	3	3	3	3
Growth	....	....	....	....
.... Glottis				
Asymmetrical	....	....	....	....
Approximation				
Inadequtae	4	2	4	2
Over	....	....	....	....
.... Vocal process				
Inflammation		2		2
.... Arytenoids				
Inflammation		2		2
Enlarged		....		....

At the time of this examination the case reported that he had occasional colds, but that he had not suffered from one for several weeks. Beyond nose and laryngeal pathologies no other significant etiological factors were found, either from our examinations or from the case histories.

The recommendations made by the staff for the treatment of this case were as follows.

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RECORD OF LARYNGEAL EXAMINATION ONE WEEK FOLLOWING VOICE CHANGE

1 2 3 4 5 Defective Normal		Right		Left	
		Anterior	Posterior	Anterior	Posterior
....	Chorda vocalis				
	Irregular	....	....	....	....
	Inflammation	4	4	4	4
	Growth	....	....	....	....
....	Thyro-arytenoideus				
	Inflammation	4	4	4	4
	Growth	....	....	....	....
5	Glottis				
	Asymmetrical	....	....	....	....
	Approximation				
	Inadequate	....	....	....	....
	Over	....	....	....	....
....	Vocal process				
	Inflammation		4		4
5	Arytenoids				
	Inflammation		....		....
	Enlarged		....		....

Three days later a final examination was made. The voice quality was definitely pleasing, and the pitch, having been lowered from F below middle C to B below low C, appeared psychologically and physiologically optimum. The following chart is a record of the final laryngeal examination. Again there was no alteration in the condition of the nose.

RECORD OF LARYNGEAL EXAMINATION MADE TEN DAYS FOLLOWING VOICE CHANGE

1 2 3 4 5 Defective Normal		Right		Left	
		Anterior	Posterior	Anterior	Posterior
5	Chorda vocalis				
	Irregular	....	....	....	....
	Inflammation	....	....	....	....
	Growth	....	....	....	....
5	Thyro-arytenoideus				
	Inflammation	....	....	....	....
	Growth	....	....	....	....
5	Glottis				
	Asymmetrical	....	....	....	....
	Approximation				
	Inadequate	....	....	....	....
	Over	....	....	....	....
....	Vocal process				
	Inflammation	....	4		4
5	Arytenoids				
	Inflammation		....		....
	Enlarged		....		....

Conclusion: This is the report of a case, 18 years of age, with falsetto voice. Possible etiological factors were nasal pathology and infection, and considerable inflammation of the entire laryngeal structure. Very shortly following the shift to normal voice the general inflammation of the vocal cords was greatly relieved, though no medical treatment had been obtained. This case suggests that inflammation of the vocal cords which may appear to be a significant primary etiological factor may actually be chiefly the result of the voice disorder, and may thus hold a much less significant part in the diagnosis.

CONSERVATION OF HUMAN RESOURCES IN WARTIME

(Continued from page 174)

INTRODUCTION, Dr. Lester B. Rogers, *Dean Summer School, U. S. C.*

TOPIC: Rehabilitation of war-disabled and physically handicapped workers in war industry.

1. An effective organization of specialists with the planned cooperation of interested agencies to secure complete rehabilitation service during and after convalescence.

2. Adjustment to the working situation as well as to community life and relationships.

3. Convalescence centers for counseling about vocational and related training.

4. New techniques in adjustment—Motion Picture: "The Doors Re-Open for The-Hard-of-Hearing."

PARTICIPANTS: Leader, William Dohr, California Bureau of Vocational Rehabilitation.

C. G. Bluett, *Calif. Vocational Rehabilitation Bureau*
Frances E. Davidson, *Teacher handicapped veterans*
A. V. Gerty, M.D., *Psychiatrist, L. A. Co. Hospital*
Victor Goodhill, M.D., *School of Medicine, U.S.C.*
Frank E. Hart, Jr., *Pres. Calif. Rehabilitation Ass'n.*
George Mangold, Ph.D., *School of Social Work, U.S.C.*
Elizabeth Payne, *School of Social Work, U.S.C.*
Robert Scott, M.D., *Veterans Admin., Los Angeles*
W. E. Smith, *Calif. Vocational Rehabilitation Bureau*
Aileen Stele, *Home Service American Red Cross*
Henry Williams, M.D., *Office of U. S. Surgeon-General*
Boris V. Morkovin, Ph.D., *Hearing Clinic: Resume*

12-2:00—Luncheon at Student Union

2-5—Afternoon panels

GENERAL CHAIRMAN, Mrs. Edythe Kennedy, *Supervisor of Special Service Dept., California Branch of U. S. Employment Service*

TOPIC. Employment of war-disabled and physically handicapped workers in war industries and civil service

1. Placement and advancement of the physically handicapped for more effective utilization of nation's manpower; removal of obstacles, meeting requirements, etc.

2. Cooperation of interested agencies

3. Training of officers and personnel for counseling, adjustment, rehabilitation and placement

4. Occupational Speech-Reading, Motion Pictures

PARTICIPANTS: Leader—John Phelps, *U. S. Employment Service*

J. F. Allert, *Representative of Organized Labor*
Armand Angel, *U. S. Employment Service, Inglewood*
Will Buzzell, *Organized Labor*
John Cornwall, *State Ins. Fund (with Douglas Aircraft)*
Ellen Mackenzie Dodson, *Welfare Coordinator, Orthopedic Hospital*

Fred Ells, *Supervisor, John Hancock Insurance Co.*
Sam Fick, *Dir. training of defense workers, State Dept.*
Charles H. Fishburn, *Man-Power Commission, L. A.*
John D. Gillis, M.D., *Industrial and traumatic surgery*
H. R. Harnish, *Labor supply commission of So. Calif.*
Glancy H. Huff, *Personnel Manager, M & M*
Doncaster Humm, Ph.D., *Industrial psychologist*
Jack E. Jones, *Emp. Mgr., No. American Aviation, Inc.*
Fenn E. Poole, M.D., *Medical dir. Lockheed and Vega*
Vern Wilson, *Vocational Consultant, U. S. Emp. Service*
B. V. Morkovin, Ph.D., *Univ. of So. Calif.: Resume*

7:00 P.M.—Banquet, in foyer of Town and Gown Club
Toastmaster: Dr. Rufus B. von KleinSmid, *Pres. U.S.C.*
Musical Program: Ann Ayers, singer; courtesy Metro-Goldwyn-Mayer Studios
Speakers and Guests:

Howard Campion, *Supt. Adult Education, Los Angeles*
Dr. Louis P. Thorpe, *Psychology Clinic, U.S.C.*
Harry D. Hicker, *Bureau of Vocational Rehabilitation*
Dr. Henry Williams, *Office of U. S. Surgeon-General*
Mrs. Spencer Tracy; Grant Mitchell; L. E. Behymer
Arthur E. Wood, *Area Supervisor, U. S. Emp. Service*

FRIDAY, JULY 3

9-12—Morning panels

GENERAL CHAIRMAN: Lillian Kositza, M.D., *Maternal and Child Hygiene, Los Angeles Health Dept.*

TOPIC: Safeguarding the Hearing of Pre-school Children

1. Education of Parents
2. Problems of Parents of Hard-of-Hearing and Deaf Children
3. Cooperation of Parents with Public Health Agencies and Pre-school Clinics

PARTICIPANTS:

Alice Chapin, *Speech Correction, L. A. Schools*
Victor Goodhill, M.D., *School of Medicine, U.S.C.*
Warren E. Griffith, *Ex. Secy., Calif. Soc. Crip. Children*
Willard B. Hargrave, *Ex. Director, Auricular Foundation*
Janet Hopkins, *Physio-therapist, Soc. for Crip. Children*
Mrs. Chas. E. Irving, *Ch'n. Except'nl. Children, P.-T.A.*
Miss F. Lukas, *Sup. Crip. Child. Serv., L. A. Gen. Hosp.*
Mrs. E. Reynolds, *American Red Cross*
Anna C. Rude, M.D., *Los Angeles Co. Health Dept.*
Edith Sappington, M.D., *Children's Bureau, U. S. Dept. of Labor*

Mrs. Richard Simon, *S. F., Exceptional Children, P.-T.A.*
Mrs. Irene Thill, *Pasa. Mothers of H.-of-H. Children*
Mrs. Spencer Tracy
Mrs. Ed J. Walker, *Pres. 10th District, P.-T.A.*

12-2—Luncheon at Student Union

2-5—Afternoon panels

GENERAL CHAIRMAN, Dr. C. C. Trillingham, *Asst. Supt. L. A. Co. Schools*

TOPIC: Conservation of Hearing Program in Schools

1. Discovery, treatment, and education of children with defective hearing
2. Effect of impaired hearing on personality, speech, reading and academic progress
3. Cooperation of all concerned with school welfare

PARTICIPANTS:

C. W. Hippler, *Dir. of Child Welfare, Pasadena Schools*
Katherine FitzGerald, *Teacher Hard-of-Hearing and deaf, Long Beach Jr. High*
Mrs. Elta Pfister, *Guidance, Burbank Schools*
Mrs. Lucy K. Proudfoot, *School Nurse, Redondo*
Mrs. Nell D. Reed, *Hard-of-Hearing and deaf in Foshay Jr. High, Los Angeles*
Mrs. Ina Sample, Jr., *High School classes for Hard-of-Hearing, Pasadena*
Jessie A. Tritt, *Supervisor of Exceptional Children, Los Angeles Schools*
Elizabeth van Arsdale, *Guidance, Ventura Schools*

5-6—Tea in Patio at Harris Hall

HOSTESSES: Mrs. Spencer Tracy, Mrs. George Wieman

WORKSHOP COMMITTEES

PROGRAM: Boris V. Morkovin, Ruth Bartlett, Mrs. Mary Rogers Miller
PROGRAM CO-ORDINATOR: Helen Scriver
PUBLICITY: Carol Warren, Mrs. Maude Braden
REGISTRATION: Mrs. Roland H. Fanton, Ruth Aus, Mrs. Alfred Peticolas
TREASURER: Mrs. Alice Beaumont, HOSTESS; Pearl Blair, Mrs. Abby Newby
SECRETARIAL: Mrs. Naomi Schultz, Mrs. Bessie Thompson
BANQUET and TEA: Mrs. Lucelia M. Moore, Mrs. Mary Jane Clifford
REGISTRATION FEE: 50 cents; Banquet, $1.25; Summary of Discussions, $1.00

Inquiries about WORKSHOP, Summer Session Courses and Hearing Clinic at U.S.C. will be answered by Mrs. L. M. Moore, 932 W. 37th St.

To reach Harris Hall, U.S.C. campus, by street car: "U" car on McClintock Ave. to West 37 Pl. or "V" car to Vermont and 37th Pl.; short walk east to No. 829.

A BIBLIOGRAPHY OF ARTICLES RELATING TO LARYNGEAL PARALYSES

By Chas. Strother, Ph.D.

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1 Acknowledgment is due to the N. Y. A. for assistance in the compilation of this bibliography.

2 Following each citation is given the date of the issue of the **Index Medicus** in which the article was listed. This is designed to facilitate a check on the citation.

(Continued to page 189)

IMPORTANCE OF SENSORY TRAINING IN SPEECH THERAPY*

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As language is normally acquired in the case of children whose hearing is normal by imitation of spoken words, it is not surprising that its development should be retarded or that speech defects should develop if a child who hears normally is handicapped with a short auditory memory span or by late maturation of the ability to discriminate between certain classes of speech sounds.

Functional sound substitutions made by children with normal hearing are composed almost entirely of consonants; these children rarely substitute one vowel for another unless they imitate a relative or playmate who makes the same sound substitution. One explanation of this fact is that plosives, fricatives, *s*, and *z* have very low phonetic power as compared with vowels. Fletcher[1] found that, if the phonetic power of unvocalized *th* is taken as 1, the average phonetic power of the fricatives *f*, *v*, and *th* is 7, that of plosives is 10, that of *s* and *z* is 16, that of *ch*, *j*, *sh*, and *zh* is 41, that of nasals is 54, that of *l* is 100, that of *r* is 210, and the average phonetic power of vowels is 430. Long *e*, the weakest of the vowels, has 220 times as much phonetic power as unvocalized *th*. One reason why *s* is mispronounced so much more often than *sh* is that *sh* has five times as much phonetic power as *s*. Some accented vowels have 3000 times as much phonetic power as unvocalized *th*. Stinchfield[2] found more frequent substitutions for *th* and for *s* than for any other English phonemes; both *th* and *s* have very low phonetic power.

A child with a short auditory memory span is likely to fail to notice speech sounds having low phonetic power and to omit these from his own speech; he returns what he hears and integrates and no more. Similarly, a child who is late in acquiring the ability to discriminate between speech sounds is likely to experience difficulty in discriminating between sounds of low phonetic power, to omit some, and to substitute incorrect sounds for others.

Durrell and Sullivan[3] state that the most common problem in teaching beginning reading rises from the fact that the child fails to remember the words which have been taught. They give as the most frequent cause of such failure lack of auditory discrimination of word elements. They found that children with excellent speaking vocabularies often fail to hear the sounds in their own speech or in that of others unless they are taught to listen for and to recognize these sounds. They maintain that the need for training in auditory perception is not confined to children with hearing defects. Of two children whose hearing is organically the same, one may have a sharp ear for word elements while the other may not notice shades of differences in sound. Children who have

*Read before the Eastern Psychological Association on April 18, 1942.

an adequate background in the phonetic element in their own speech usually learn to read very rapidly.

Plummer[4] compared the ability of 393 Louisiana State University undergraduates to discriminate plosives, fricatives, and sibilants with their ratings on a 6B Western Electric audiometer test and found that neither mild nor severe losses in hearing in the high-frequency range could account for any appreciable difficulty in discriminating between these consonants. He found no relationship between hearing losses at specific frequencies and ability to discriminate between specific consonants. He found that plosive-fricative pairs were missed 5% more often than pairs of plosives, that pairs of fricatives were missed 29% more often than plosive-fricative pairs, that voiceless consonants were missed slightly more often than voiced consonants, and that final consonants were missed more often than those in any other parts of words.

Linton's[5] experiments at Stanford University showed little if any relationship between speech sound discrimination and intelligence, and only a slight relationship between speech sound discrimination and length of auditory memory span as tested by vowel sounds.

West[6] maintains that one of the important factors in the development of speech is the ability to hear and to distinguish faint speech sounds of high pitch. Although failure or delay in developing this acuity and discrimination ability is sometimes caused by disease of the ear, it is frequently due to a natural slowness of maturation of this special faculty, which often seems to be a family trait.

Tavis[7] gave 300 pairs of consonants combined with a constant neutral vowel and 66 pairs of vowels to 383 normal speakers and to 186 speech defectives and found that at every compared age level individuals with mild functional speech disorders of articulation made significantly more errors than did the normal speakers, and that those individuals manifesting the severer speech disorders gave a relatively high relationship between the sounds mispronounced and those not discriminated on the test.

The writer has been trying for many years to develop a satisfactory test for acquiring accurately the relative ability of young speech defectives to discriminate between different classes of speech sounds of different pitch frequencies. He began by testing vowels, cognates, high-frequency consonants, and a group of miscellaneous consonants not included in the other groups. It was found that vowels were discriminated correctly by sound substituters 75% of the time, the miscellaneous consonants 65%, the cognates 61%, and high-frequency consonants 59% of the time. Half of the children missed more than half of the high-frequency consonant changes.

The writer then further divided the groups into vowels, semi-vowels (*m, n, ng; l, r, w,* and *y*), pressure changes made from approximately the same mouth mold (*t* for *s, ch* for *sh,* etc.), cognate changes (confusion of voiced and unvoiced consonants made from identical mouth molds as *t-d, s-z*), high-frequency plosives, and high-frequency fricatives and sibilants.

As most young children are bored by nonsense syllables and refuse to repeat these after the examiner, it was necessary to use real words,

most of which are contained in "A Reading Vocabulary for the Primary Grades" by Arthur I. Gates. Pairs of the more familiar words of approximately equal familiarity were used at the beginning of all tests; more difficult words had to be used toward the ends of tests in the case of the rarer speech sounds. The pairs of words to be discriminated were exactly alike save for the change of a single speech sound at the beginning, middle, or end of the word (*th*ing-*s*ing, ca*t*-ca*p*).

The first test[8] consisted of reading to the child three one-syllable words at the rate of approximately one word per second, pausing for three seconds, and reading in the same manner this list of words with the other member of the pair substituted—(shall, far, *th*ing-shall, far, *s*ing). The series to be tested consisted of an equal number of changes at the beginnings and at the ends of words, and in the first, second, and last words read. To make sure the child understood the test, he was given a trial series beginning with very noticeable changes and increasing in difficulty to the level of the word-pairs of the test itself. When fatigue set in the test was discontinued for the day, and resumed at the child's next visit to the clinic. This test was found too difficult for the average speech defective under the age of seven; hence a simpler test was devised for the younger children, who constituted a majority of the sound substituters.

The second test contained one member of the word-pair to be discriminated twice and the other member once—(*th*ing, *th*ing, *s*ing). The examiner read the three words to the child in a monotone at the rate of one per second and asked the child to tell whether the first, second, or last word was different from the other two words. The same preliminary test was given and the same precautions taken as on the first test. Although the children understood the second test much better than the first test, it still proved too difficult or too boring to several of the younger children.

The third test[9] consisted of attractive black and white sketches of the objects whose names are to be discriminated (ca*t*-ca*p*). Six pairs of such pictures were laid out in random order on a table of comfortable height in front of the child and he was asked to point to each picture as its name was spoken. He was given a score of 1 if he pointed to the right picture the first time it was named, a score of 0.5 if he pointed to it the second time it was named, but failed the first time, and a score of 0.3 if he succeeded on the third trial but failed on both the first two trials. Only three trials were given. The child was taught the names of all twelve pictures presented at one time before the test was begun. This third test was a success from the start. The children considered it a game which was lots of fun; hence it was not difficult to motivate them or to get them to concentrate consistently.

The percentage of word-pairs correctly discriminated on a given test was obtained by dividing the total raw score obtained on that series by the number of word-pairs tested on the first two tests, and by twice the number of pairs tested on the third test. A comparison of these percentages on the different series of tests shows whether a child is relatively weak in discriminating vowels, semi-vowels, pressure changes, cognates, plosives, or fricatives, and enables the speech therapist to

give the child further drill on those types of word-pairs which he failed to discriminate, without wasting time in drilling him on those types of changes which he usually discriminates.

Sensory training consists simply in asking the child to keep on discriminating more of these word-pairs of increasing difficulty until he has learned to hear all parts of words accurately.

Although the auditory memory span for digits has been well established as 3 at age 3, 4 at age 4, 5 at age 7, and 6 at age 10, that for phonemes and syllables has never been so standardized.

West[6] asserts that auditory memory span is probably one of the most important factors in determining the time of onset of speech and its rate of maturation. He states that the child of three should be able to recall two sounds, the child of eight between two and three sounds, and the adolescent child and the adult should average well above three sounds. He seems to have tested the auditory memory span for plosives, fricatives, and sibilants which other writers have found have a shorter memory span than vowels, due partly to the low phonetic power of these consonants and partly to difficulties in sound discrimination. West advises against using the auditory memory span for vowels and semivowels because their phonemes are not sufficiently definite, and against mixing voiced with voiceless sounds.

Fontaine[10], one of West's students, found that the norms for auditory memory span for vowels is substantially lower than that for digits; that, although there was only a low correlation between memory span and age and intelligence, memory span tended to increase until age 9; and that the speech defective group as a whole did not differ from an equal control group of 200 children in auditory memory span for sounds to a significant degree.

Metraux[11] found vowels (not consonants) a valid test for auditory memory span for speech sounds because vowels can be easily reproduced by children; they have adequate phonetic power to be readily heard, and have little associational value. Thirty-four of her speech defectives from the Ann Arbor, Michigan, schools gave an average auditory memory span for vowels of 2.15 compared with an average of 1.82 for normal speakers; but these children gave an average auditory memory span for consonants of only 0.24 and 1.21, respectively. She found that speech defectives with articulatory defects did slightly better with vowels, whereas stutterers and those with voice defects did slightly better with consonants. Audiograms did not show a hearing loss significant enough to affect the results, or to account for the lower score on the high-frequency consonants. She found that auditory memory span showed no increase with any degree of conformity along with increase in age or in intelligence rating. Her subjects consisted of 25 boys and 9 girls in both the speech defective and the control groups. Their ages ranged from 6 to 10 years, and their intelligence quotients from 70 to 130.

The writer has tested the auditory memory span for digits, for phonemes, and for syllables in 150 speech defectives who attended ten local speech clinics and found that the span for phonemes and for syllables increases consistently with age until about age 12, and remains

very close to the standard auditory memory span for digits when phonemes and syllables are tested in real words. Diphthongs *ch, j, sh, th,* and *zh* seem to count as single sound units, and *x* as two sound units, *ks.* Silent letters should not be counted in determining auditory memory span; thus the word *though* is composed of but two sound units.

Some young children with an auditory memory span for three digits or less seem to fail on consonant blends in all positions; thus they can say *cat,* but not *ask* or *spy.* A few children with a span for four digits or less frequently fail on groups of three adjacent consonants as *street* and *sixth.* This trouble is probably due to late maturation of the ability to discriminate between similar sounds of low phonetic power, because most of the children with identical spans returned such words correctly. The auditory memory span for phonemes was found to be slightly higher than that for digits, and that for syllables slightly higher than that for phonemes. The word method of testing auditory memory span seemed a much more practical manner of testing speech sounds in speech defectives than nonsense series of phonemes which are seldom understood and never used by young children. (Half of the children tested had not attained their seventh birthday.)

The following table contains the average auditory memory span for digits, for phonemes, and for syllables for speech defectives who substituted one sound for another, who omitted phonemes, and who were late in learning to speak. These spans are given at the different ages for which the conventional auditory memory span for digits remains constant.

	AGES		
	4-6	7-9	10-13
SOUND SUBSTITUTIONS (81 subjects)			
Span for digits	4.2	5.4	5.9
Span for phonemes	5.0	5.5	6.4
Span for syllables	5.6	5.9	6.7
ELISIONS (76 subjects)			
Span for digits	4.1	4.8	5.3
Span for phonemes	4.2	4.7	5.3
Span for syllables	4.5	5.5	5.6
DELAYED SPEECH (33 subjects)			
Span for digits	3.7	5.6	4.8
Span for phonemes	4.0	5.4	5.5
Span for syllables	3.9	5.9	5.7
NUMBER OF SUBJECTS	70	44	29

Although most of these children made sound substitutions, only those were included in the sound substitution group whose speech defects were not complicated with other speech defect symptoms. Many of the children who were late in talking omitted sounds; such children were included in both elision and delayed speech groups.

Short auditory memory spans were found in 13% of the sound substituters who manifested no other speech symptoms, in 33% of those who omitted phonemes, and in 45% of those who were late in acquiring speech. Short auditory memory span is probably the cause of many cases of elision and of delayed speech as indicated on an earlier study.[12]

A child who is handicapped with a short auditory memory span can be taught to build up words which normally require a longer memory span from series of larger units which comes within his memory span. For example, monosyllabic words and syllables which contain too many phonemes can be built, like the word *street, from eat* through *tr-eat, treat, s-treat,* to *street,* pausing between sounds or groups of sounds as indicated. Although most children could build up the word *Indian* at once from its three syllables, *In-di-an,* they would have to build up the name of the city, *Indianapolis,* from such larger units as *Indian-apple-iss.* Children with short auditory memory spans should be drilled as far as possible in short words of intrinsic interest which come within their memory spans, and contain no consonant blends. Parents, teachers, and playmates should thus limit their own vocabularies when speaking to children with short auditory memory spans, and pronounce these words very slowly and distinctly.

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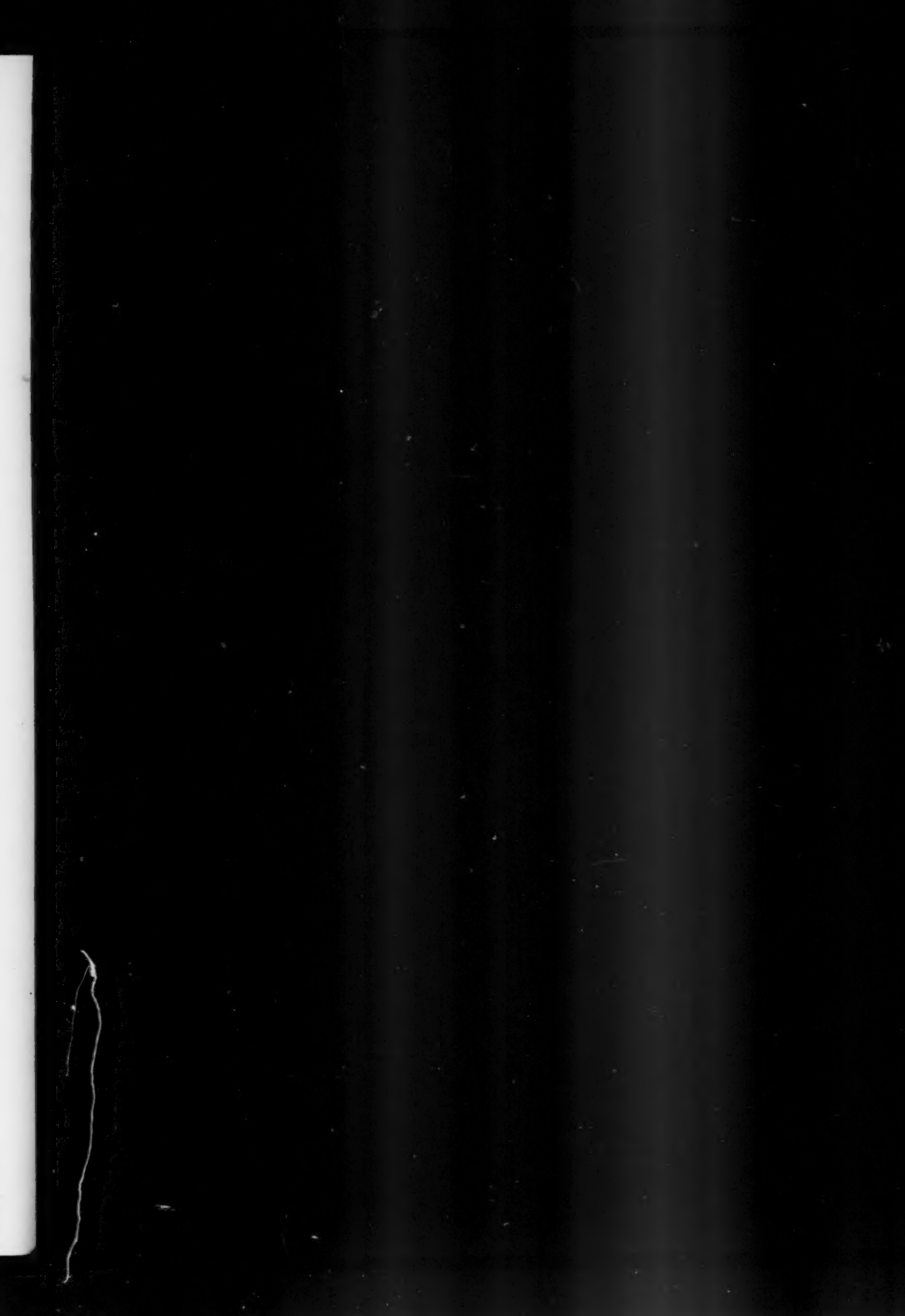
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